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THE STATIC AND KINETIC SYSTEMS OF MOTILITY

PRESIDENTIAL ADDRESS

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NEW YORK

The subject of motility is one of great interest to scientific men in many fields of investigation. Neurologists, particularly, have always had a peculiar interest in motor function and its disorders, and to neuropathology must be credited much that we know of the underlying neural and muscular mechanisms.

During the last half century our knowledge of this important function of the living organism has made great progress and each decade has marked some new advance in the realm of motion, as well as in the sensory sphere. For these two great functions of the living organism are the pillars on which have been reared those marvellous mechanisms of the central nervous system, the phylogeny of which is the story of all organic life.

The problem of motion and sensation must, therefore, ever remain a living issue in the realm of science, as life itself traverses these two pathways side by side in the slow and imperceptible processes of evolution.

It would carry us too far afield at this time to record the achievements of the many investigators whose names are imperishably linked with the history of motility. This I shall hope to do later when my subject is presented in more extended form. I wish, however, to acknowledge having made free use of this vast accumulation of facts in the conception of the efferent nervous system which I am about to formulate.

This hypothesis is not concerned so much with the elusive intricacies of motility itself, as with the dual nature of its underlying systems.

COMPONENTS OF MOTILITY

I believe that motility consists of two components, each being represented throughout the entire efferent nervous system by separate neural mechanisms, which are mutually cooperative and yet physiologically and

*Read at the Forty-Sixth Annual Meeting of the American Neurological Association, New York, June, 1920.

anatomically distinct. This is true not only of the segmental nervous system but also of the larger integrating and coordinating masses of the brain and cerebellum. Under normal conditions they are inseparable and act in unison, and the harmony of their relation is only disturbed by the dissociation of accident or disease.

One of these components of motility is the movement proper, which is subserved by the *kinetic system.*

The other represents the more passive form of contractility, which we recognize in tonus, posture, attitude, and equilibrium. This static function of the efferent system is subserved by separate neuromuscular pathways, which may be termed the *static system.*

The term static is used here in the same sense as in the physical sciences, where it relates to the laws governing matter at rest and the forces producing equilibrium. I have found no other word which expresses better my conception of this other great function of the efferent system.

THE PHYLOGENY OF CONTRACTILE TISSUE

Before proceeding to a presentation of the evidence on which this conception is based, I would first ask your indulgence while I review briefly the phylogeny of the nervous system in its bearing on the development of movement.

All of those more complicated muscular activities which we study in man have, like every other function of the human organism, a very humble origin. And in order to detect the earliest manifestations of motility we must retrace our steps to the lowest rung of the ladder of life, where the ameboid movements of unicellular organisms allow these minute beings some slight increase in the range of their activities. Even in this lowly form the slow plastic movement is followed by a faint postural or static effort, when the pseudopodium of the cell becomes fixed to some neighboring object by molecular attraction or adhesion. The tiny organism then not only reaps the advantages of movement but the equally valuable effect of postural fixation under new conditions.

For if the power to move in space were not associated with an equal ability to control such movements in terms of posture, attitude and equilibrium, the advantages of motility would be of very doubtful value to the higher organisms.

The next advance in kinetic evolution is the ciliated movement. Here a protoplasmic cell has projecting from its free surface minute fibrils which are derived from the intracellular network of the organism. These cilia are capable of quick rhythmic, to and fro movements quite unlike the slow, plastic transformations of structure which characterize the first protoplasmic efforts of the ameba in the realm of movement.

As one ascends the scale of life, from one phylum to another, there is developed in the course of evolution a more complex contractile substance, the muscular tissue, which is the active agent in producing that great diversity of movement which characterizes animal life. The first development is the unstriped muscle which, according to some authorities, is present as low in the scale as sponges (Parker [1]), and is constant in all higher forms. The quality of its movement is slow and plastic. Striated muscle, which represents a higher differentiation of contractile tissue, makes its appearance in worms and mollusks, and constitutes the skeletal musculature of vertebrates. The quality of its movement is quick and elastic.

A very striking illustration of the dual nature of muscle function may be observed in certain of the invertebrates. In bivalved mollusks closure of the shell is affected by the contraction of two distinct muscles, a contractile muscle which is striated, and an arrest muscle which is nonstriated. The rapid movement of closure of the shell is produced by the striated muscle. This is accompanied by a slower contraction of the arrest muscle which becomes firmly fixed as soon as the movement ceases. The arrest muscle thus fixes in posture the closing movement of the shell, which is then able to withstand an enormous degree of pressure before relaxation is induced. A similar division of movement and posturing muscles may also be observed in Echinus and Pecten and these have separate and distinct sources of innervation (von Uexküll [2]).

Comparative anatomy shows clearly that the muscular system, like all other structures of the body, is in a state of evolution, adapting itself to the changing conditions of the organism. Development is from the slow movement of the unstriped to the quick movement of the striated muscle fiber. And in man all gradations may be observed from the lowest type of unstriped muscle to the highest type of striated muscle fiber.

Striated muscle contains two substances, both capable of contraction. The one is the disk mechanism of the muscle fiber, which gives rise to the quick movement or the twitch. The other is the sarcoplasm which yields a more plastic form of contraction. It is, I believe, not unlikely that the simple protoplasmic movements of the ameba and the quick rhythmic waves of the cilia are the prototypes of the more elaborate functions of striated muscle.

Marshall,[3] after an exhaustive study of the muscles of vertebrate and invertebrate life, has reached certain important conclusions. He

1. Parker: The Reaction of Sponges, J. Exper. Zool. **8**:39, 1910.

2. von Uexküll: Studien über den Tonus, Ztschr. f. Biol. **44**:269, 1903; ibid. **58**:305, 1912.

3. Marshall, C. F.: The Structure and Distribution of Striped and Unstriped Muscle in the Animal Kingdom and a Theory of Muscular Contraction, Quart. J. Microsc. Sc. **27**:75, 1888.

believes that the disk mechanism of the muscle fiber is the final product in course of evolution from the primitive intracellular network of the cell. This is represented in the unstriped muscle fiber by the longitudinal fibrillations. He traces from its origin the gradual development of the contractile mechanism and shows its relation to the quality of movement. A movement is slow or quick according to the quality and degree of differentiation of this constituent of the muscle fiber.

A clear conception of the phylogeny of contractile tissue is important to my theory as I do not regard the skeletal muscle mass in man as a mere aggregation of muscle fibers of a standard type, but rather as systems of end organs representing different stages of phylogenic development, and thus corresponding in some measure to similar differentiation in nerve tissue.

I believe that the highest functions of motility are carried out by more highly developed muscle fibers than are the lower forms of movement. For the functions of the efferent nervous system and the muscular system are so closely intertwined that one can scarcely conceive of evolutionary progress in one without corresponding changes in the other, to meet the demands of more elaborate functional activity.

TYPES OF MOTILITY AND POSTURE

Let us now leave the question of muscle tissue and consider, for a moment, the general types of motility in animal life. These are usually divided by physiologists into three groups, reflex, automatic and associated, and isolated synergic movements.

It should be remarked, however, that one group merges imperceptibly into the other and the transition is so gradual that it is difficult to say where one type ceases and the other begins. For as Herbert Spencer pointed out, the progress of evolution is from the most automatic to the least automatic and cessation of automatic activity and the dawn of volition are one and the same thing.

In the lowest forms of life, movement and posture are of a purely reflex character and depend on a primitive reflex arc, which is the functional unit of the nervous system. In higher forms these reflex units are combined and more complicated reflex activities result. These in turn are gradually brought under the control of still higher integrating and correlating centers with corresponding advances in the variety and forms of movement.

There are many extraordinary evidences of reflex action in the lower forms of life, both in the realm of posture and of movement. There are such movements as the reflex of defense, the flight reflex, the turning reflex, that strange reflex of self-mutilation by which an extremity is discarded to escape a threatened danger. There is a disguise reflex characterized by alteration of color, also a tonic reflex

producing a general rigidity and one simulating the posture of death, and many others that have been acquired in the struggle for existence.

In the "automatic and associated group" there are movements of crawling, walking, running, jumping, swimming and flying. These movements are usually bilateral and rhythmic, or alternating and rhythmic, and find their highest expression in vertebrates. All of these movements, reflex as well as automatic, have their corresponding static components, and every cessation of movement is immediately followed by fixation of the contraction in posture.

In the higher mammals and especially in man one encounters a new form of movement which differs from reflex and automatic movements in the quality of individuality and dissociation. Thus, one of the extremities which in reflex and automatic movements plays a fixed rôle as part of a mechanism, becomes capable of isolated and unilateral effort under voluntary control. This form of movement represents an immense advance in the evolution of motility and underlies the development of all skilled movements which have reached such extraordinary perfection in man.

In all of these isolated forms of voluntary movement there is also the element of posture, and it may be stated without fear of contradiction that there is no form of motility from the simple reflex to the most skilled and individualistic type of movement which functions without a corresponding static mechanism.

EVOLUTION OF THE NERVOUS SYSTEM

With the gradual increase in the range of movement and posture in organic life there has been a corresponding development of nerve structure, for every movement is represented as such in the mechanism of the nervous system. As one ascends the animal scale there is an increasing complexity in the arrangement of the central nervous system, due to the development of higher and higher levels of coordination and control. In man and the primates the original simplicity of the segmental neuraxis is quite concealed by the enormous development of cerebrum, cerebellum, and their respective projection systems in the spinal cord.

Hughlings Jackson,[4] following the philosophic teachings of Herbert Spencer, was the first to grasp the full significance of evolutionary principles as applied to the central nervous system of man. His conception of functional levels, each higher level controlling the activities of the lower centers, stands today, and will, to my mind, continue to dominate all conceptions of nerve functions, psychic as well as

4. Jackson, Hughlings: Croonian Lectures, Brit. M. J. 1:660, 703, 1884.

somatic. For in the central nervous system of man is written in terms of functional levels the whole history of the evolution of organic life.

In a general way the central nervous system may be said to represent three great structural and functional divisions. There is a segmental nervous system, which contains the great reflex systems of the neuraxis, a paleo-encephalon which represents the essential sensory and motor mechanism in lower forms, and a neo-encephalon which has its greatest development in man, and subserves the highest functions of motility and sensibility.

It is my belief that these three great functional divisions of the nervous system are related to the three great types of movement and posture enumerated in the foregoing.

In this conception, I regard the cerebellum as the great correlating center for the static functions of motility, in contrast to the corpus striatum and Rolandic area, which I regard as the essential correlating centers for the control of their respective forms of kinetic function. Furthermore, I believe that these systems are represented by special end organs in the skeletal muscles in the same manner as we conceive sensations with their special systems and modes of termination.

THE AUTHOR'S HYPOTHESIS

I will now proceed to a presentation of the evidence on which my hypothesis is based, which, however, on the present occasion, cannot be given in detail but can only be outlined in a very general way. Let us first consider the muscular system, which contains all of the contractile end organs of the efferent mechanism.

The skeletal musculature is composed of striated muscle fiber held together by connective tissue, by means of which the contractile mass performs its functions. The purely anatomic divisions of muscles are of comparatively little importance in the physiology of motion. For as Hughlings Jackson pointed out, "movements not muscles" are represented in the nerve centers and usually a number of muscles participate in any given movement.

We must, therefore, conceive the efferent nervous system, including the muscles, as organized for the execution of a great variety of movements which become more and more numerous and complex as we ascend the scale of life.

As we have seen, each striated muscle fiber is composed of two distinct substances which are now generally recognized by physiologists as subserving different types of motor function. In a general way it is estimated that the disk system constitutes about from one sixth to one half of the total muscle mass, the remaining portion of which is sarcoplasm.

DUAL INNERVATION OF MUSCLE FIBERS

Each muscle fiber contains a motor nerve ending, which is the terminal of a medullated nerve fiber. For many years this was thought to be the sole innervation of the muscle fiber, until Perroncito [5] and Boeke [6] demonstrated the existence of another smaller nerve ending in the striated muscle fiber which was shown to be the terminal of a non-medullated nerve.

Boeke expressed the opinion that the motor end plate of the medullated nerve fiber furnished the stimulus for the disk system while the accessory sympathetic terminals were in control of the sarcoplasmic portion of the contractile mechanism, a conception which Mosso had previously expressed in his interpretation of the tonus and twitch components of muscle function. Boeke carried his investigation still further, and in order to prove the independent nature of these two systems, he cut the trochlear nerve in the cat and studied the nerve degenerations in the superior oblique muscle. All of the motor end plates in connection with the medullated nerve fibers were found in various stages of disintegration while the accessory sympathetic nerve endings showed no evidences of degeneration.

One could hardly offer better evidence of the independence of these two systems of nerve endings.

These histologic studies awakened considerable interest in the physiologic world, as Botazzi [7] had previously enunciated a theory of dual contractility for the muscle of the batrachian heart. In this hypothesis the slow tonic contraction of the sarcoplasm supported and reinforced the quicker movement of the disc system.

It is also interesting that physiologists have detected certain differences in the metabolism of muscles which are probably related to these two forms of activity of the muscle fiber.

Pekelharing [8] demonstrated the existence of two chemical processes in muscle metabolism. One was related to the disk system, and was characterized by the utilization of nonnitrogenous substances. The other was concerned with the utilization of albuminates and this was possibly dependent on the chemical activity of sarcoplasm. Other experiments have shown that the expenditure of energy of the active

5. Perroncito: Etudes ultérieures sur la terminaison des nerfs dans les muscles à fibres striées, Arch. ital. de Biol. **38**:393, 1902.

6. Boeke: Die motorische Endplatte bei den höheren Vertebraten, Anat. Anzeiger **25**:193, 481, 1909.

7. Botazzi: The Oscillations of the Auricular Tonus in the Batrachian Heart with a Theory on the Function of Sarcoplasm in Muscular Tissues, J. Physiol. **21**:1, 1897.

8. Pekelharing: Die Kreatininausscheidung beim Menschen under dem Einfluss von Muskeltonus, Ztschr. f. physiol. Chem. **75**:207, 1911.

muscle contraction (tetanus) is far greater than is that of posturing muscle (tonus). This explains the relative unfatigability of the posturing or sarcoplasmic function of muscle. It is certainly significant that muscle fibers consisting of two types of contractile substance should also present two distinct forms of chemical activity.

Another striking confirmation of a dual efferent system is to be found in the researches of Kure, Hiramatsu and Naito,[9] on the innervation of the diaphragm. These investigators were able to show by animal experimentation that the phrenic nerve conveys the kinetic impulses for this important muscle, while its tonus is received through the splanchnic system of the sympathetic. If the phrenic nerve is cut, the diaphragm is paralyzed but tonus is preserved. When the sympathetic nerve is cut, tonus is lost but motility is unaffected. As the diaphragm performs a relatively simple function it is not surprising that these two systems are dissociated and, as had already been shown by experiments of Boeke, the same is true for the kinetic and static systems of the ocular muscles.

The attempt has also been made to demonstrate a similar dissociation in the innervation of the lower extremities by cutting the trunk of the abdominal sympathetic. This experiment was performed by Dusser de Barenne[10] who claims to have produced a demonstrable loss of tonus in the corresponding muscles. These results, however, have not been confirmed by subsequent observers (Cobb,[11] Beritoff,[12] and others) and it appears probable that the sympathetic efferent system of the extremities takes a different route, not passing by way of the vascular system.

As the extremities represent a later phyletic addition to the muscular system, it is possible that the efferent static innervation for these structures may pass to the muscles with the motor nerves. In this connection it is interesting to note that Ransom[13] has shown the existence of a large complement of nonmedullated nerve fibers in peripheral nerves. Further investigation will be necessary, however, to

9. Kure, Hiramatsu and Naito: Zwerchfelltonus und Nervi splanchnici, Ztschr. f. exper. path. und Therap. **16**:395, 1914.

10. Dusser de Barenne: Ueber die Enthirnungsstarre (Decerebrate Rigidity) in ihrer Beziehung zur effereaten Innervation der quergestreiften Muskulatur, Folia neuro-biol. **7**:651, 1913.

11. Cobb: A Note on the Supposed Relation of the Sympathetic Nerves to Decerebrate Rigidity, Muscle Tone, and Tendon Reflex, Am. J. Physiol. **46**:478, 1918.

12. Beritoff: Die tonische Innervation der Skelettmuskulatur und der Sympathicus, Folio neuro-biol. **8**:421, 1914.

13. Ransom: Unmyelinated Nerve Fibers as Conductors of Protopathic Sensation, Brain **38**:381, 1915.

clarify this question and to show any relationship between the non-medullated fibers described by Ransom and the sympathetic terminals of the striated muscle fiber.

The existence of two separate contractile systems in striated muscle fiber is also shown very clearly by the electromyogram. In myotonia congenita, Gregor and Schilder [14] have demonstrated the presence of two distinct contraction waves, the one quick and referable to the disk system, the other slower, more sustained, and of sarcoplasmic origin. A similar dissociation of quick and slow muscle waves may also be produced in the electromyogram of veratrin and creatinin intoxication.

PERIPHERAL MECHANISMS OF TWO SYSTEMS OF MOTILITY

In the theory which I am endeavoring to unfold the disk is to be regarded as the end organ of the kinetic system, and the sarcoplasm as the end organ of the static system, just as we conceive the heat and cold spots or the tactile corpuscles as end organs in their respective sensory spheres. These end organs of the skin have undergone special differentiation on certain parts, and have thus acquired a greater accuracy and refinement of function.

I believe that a similar specialization of function and structure exists in skeletal muscles. By this I would emphasize what perhaps is not generally recognized, that the muscle fiber system, like the nerve fiber system, participates in phyletic differentiation, and that those muscle fibers which subserve the higher functions of neokinesis are more highly specialized than are those which are concerned with paleokinetic function.

According to this conception the corticospinal system and the striospinal system would both be represented by special contractile end organs.

As the striospinal system is older, the muscle fibers subserving this function would be less highly differentiated than those subserving the highest cortical function. Let us see what facts may be derived from histology, and physiology, in support of this point of view. As long ago as 1874, Ranvier pointed out that in muscles there are two kinds of fibers, which he described as pale and dark fibers. The pale fibers are rich in contractile substance and contain comparatively little sarcoplasm. The dark fibers are rich in sarcoplasm with a relatively simple disk mechanism. Both kinds of fibers are found in all muscles in varying degrees.

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14. Gregor and Schilder: Zur Theorie der Myotonia, Neurol. Centralbl. **32**:85, 1913.

The pale fibers are the quick contracting, and the dark fibers the slow contracting fibers of the physiologists (Pankul[15]). And the quickness of a muscle contraction which varies considerably in different portions of the body depends on the relative number and distribution of these two types of fibers. It has also been shown that those muscles which are subjected during the life of the organism to the most constant activity are especially rich in sarcoplasm (Knoll[16]).

I believe, therefore, that the more highly differentiated pale fibers are related to the neokinetic system, while the dark fibers are concerned with paleokinetic motility. As the evolution from the lower to the higher movement is gradual, there are transition forms between these two types of fibers.

The skeletal muscles may therefore be regarded as contractile tissue, which bears the imprint of the phylogenetic struggle in the same measure as the nervous system which controls it. Considered as a whole it is the end organ of the efferent system and in its mass are represented the great kinetic and static pathways of the brain and cord.

THE SEGMENTAL NERVOUS SYSTEM AND THE DUAL FUNCTION OF MOTILITY

Let us now consider the segmental nervous system in its relation to a dual function of motility. At this level are represented the earliest centers for movement and posture, and the relative simplicity of its reflex mechanisms has made it an especially attractive field for experimental study. Sherrington,[17] who is the acknowledged authority on the various reflex activities of the nervous system, has reached the conclusion that two types of reflex action may be recognized. There is a reflex movement which he calls the phasic reflex, and a reflex posture which he terms the tonic or postural reflex, and in the realm of reflex action he indicates very clearly the duality of function which I would extend to the whole of the efferent system.

He also has shown in his study of "postural activity of nerve and muscle" that coordination and reciprocal innervation are as much a part of the tonic, or postural, reflex as they are of the phasic reflex mechanism. His experiments on decerebrate rigidity, as well as those of Magnus and Kleijn,[18] show conclusively that innervation and coordination are as important in the realm of posture as in that of movement.

Reflex action is therefore dependent on a peripheral kinetic system which terminates in the disk contractile mechanism. And reflex posture

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15. Pankul, in Luciani: Textbook of Physiology **3**: Chapter 1.

16. Knoll: Ueber Protoplasmaarme und Protoplasmareiche Muskulatur, Denkschr. d. k. Akad. d. Wiss. z. Wien. Math. naturw. Kl. **58**:633, 1891.

17. Sherrington: Postural Activity of Muscle and Nerve, Brain **38**:191, 1915.

18. Magnus and Kleijn: Die Abhängigkeit des Tonus der Extremitäten muskeln von der Kopfstellung, Pflüger's Arch. f. d. ges. Physiol **145**:455, 1912.

is dependent on a peripheral static system which terminates in the sarcoplasm of the muscle fiber. These are the neuromuscular representatives at the segmental level of the kinetic and static pathways of motility.

One function of the peripheral static system is the so-called "lengthening and shortening reactions" of muscles. This intrinsic function of muscle tissue is dependent on postural tonus, by which a muscle may be lengthened or shortened during contraction without corresponding changes of tension. This alteration in postural length without corresponding changes of tension facilitates the easy transition from posture to movement which characterizes motility.

Sherrington has also shown the great importance of this posturing mechanism in hollow viscera, like the bladder, the postural tonus of the muscular walls of this organ allowing considerable changes of volume without increase of the intravesical tension. It has also been definitely established that a similar mechanism exists in the vascular system and in other structures under the control of the autonomic nervous system.

Before leaving the peripheral systems of motility, some mention should be made of the "all or nothing" principle of Bowditch [19] which apparently governs all forms of muscular contraction. His experiments were carried out on the heart muscle of the frog and showed that an induction shock produces a muscle contraction or fails to do so according to the strength of the current. If a contraction is induced, it is the greatest of which the muscle fiber is capable, and there are no gradations with varying strengths of current.

Keith Lucas [20] has shown that this principle also applies to the skeletal muscles. Accordingly, the strength of a muscular contraction is not dependent on the strength of the current, but on the number of muscle fibers participating in the contraction. The refinements and gradations of movement are therefore dependent on the number of muscle fibers participating, and not on variations in the strength of the nerve impulse.

CENTRAL PATHWAYS OF TWO SYSTEMS OF MOTILITY

Let us now leave the peripheral mechanisms and pass to a consideration of the central pathways of motility.

In a previous study of paralysis agitans,[21] I expressed myself as favoring the conception of two great motor systems from brain to

19. Bowditch, in Bayliss: Textbook of Physiology, 1919, p. 383.

20. Lucas, Keith, in Bayliss: Textbook of Physiology, 1919, p. 383.

21. Hunt: Primary Atrophy of the Pallidal System: A Contribution to the Functions of the Corpus Striatum, Brain **40**:58, 1917; Arch. Int. Med. **22**:647, 1918.

muscle, one subserving paleokinetic and the other a neokinetic function. The corpus striatum, according to this view, is the paleokinetic center for the control of "automatic and associated movements," and the Rolandic area is the neokinetic center for "dissociated movements" of cortical origin. Their static functions I believe are subserved by other pathways.

It should be remarked, however, that the thalamic portion of the paleo-encephalon is closely associated with the cerebral cortex by afferent and efferent tracts, by means of which paleokinetic motility may be directly controlled from the cortical sphere (cortico-thalamo-striospinal system). This higher control of the striospinal system makes possible a finer differentiation of paleokinetic motility and explains the occurrence of purely involuntary movements of cortical origin. It also bridges the gap between the striospinal and cortico-spinal types of movement and renders the transition from the older to the newer forms of motility a more gradual process.

In striking contrast to the paleokinetic system is the neokinetic system. This is a direct pathway from the cerebral cortex to the spinal cord for the control of isolated synergic movement. It takes its origin in the motor cells of the Rolandic area and passes by way of the pyramidal system directly to the anterior horn cells of the spinal cord, and from there to a special disk representation in muscle subserving the dissociated movements of cortical origin.

These two great motor systems, the pyramidal and extrapyramidal, according to this theory, are both represented in nerves and skeletal muscles. They are in a sense complementary and their independence is only unmasked by the dissociations of disease.

A critical analysis of the symptomatology of pyramidal and extrapyramidal disease (spastic paralysis and paralysis agitans) are strongly corroborative of this hypothesis (Hunt [22]).

The central pathways for the control of the static component of motility are, I believe, distinct from those subserving its kinetic function. The essential suprasegmental mechanism for the control of static motility I believe to be the cerebellum. The importance of this organ in the regulation of muscle synergy and tonus and in the maintenance of equilibrium has been recognized by all observers, and yet, notwithstanding years of patient investigation, a certain obscurity still clouds our conception of its function and relation to motility.

DeBoer [23] has recently considered it as the central mechanism for the control of reflex tonus and the sarcoplasmic function of the muscle

22. Hunt: The Existence of Two Distinct Physiological Systems for the Transmission of Motor Impulses in Peripheral Nerves, Brain **41**:302, 1918.

23. de Boer: Die Bedeutung der tonischen Innervation für die Funktion der quergestreiften Muskeln, Ztschr. f. Biol. **47**:239, 1914.

fiber, and has presented an imposing array of evidence in favor of this hypothesis. Tilney,[24] in a comprehensive study of the cerebellum, has enlarged our conception of its synergic function to include the synergic unit. This is represented by a single muscle and its antagonist which contains a "dominant and check mechanism" capable of kinetic and akinetic phases. In this conception both the kinetic and static functions are subserved by the unit of synergy.

While accepting in the main the classic conception of cerebellar synergy as enunciated by Babinski, I would limit this function to a control of the static synergies of motility. The kinetic synergies are controlled by their respective kinetic mechanisms.

Anatomically, the cerebellum has close connections with both the cerebrum and the spinal cord. It receives impulses directly from the spinal cord, which pass to its central portion (vermis), as well as impulses from the cerebral cortex, which pass by way of the pons varólii to the cerebellar hemispheres.

In the cerebellum there are phylogenetic evidences of two great systems, which correspond to similar divisions of the cerebrum. There are a paleocerebellum, or vermian system, and a neocerebellum or hemispheric system.

The efferent cerebellar systems originate in the central nuclei (the dentate, nucleus globosus, emboliformis and fastigii) and traverse the superior cerebellar peduncles.

The older cerebellum stands in relation to what may be termed the *paleo-static system* which controls the static functions of movement of the "automatic and associated type." It takes its origin in the older nuclei of the vermis cerebelli.

The cerebellar hemispheres, on the other hand, regulate the higher postural functions of motility through the medium of a *neo-static system,* which originates in the cells of the dentate nucleus. This is controlled from the cerebral cortex by a special system of fibers passing from the frontal lobe to the pons varolii and thence to the opposite hemisphere of the cerebellum. The frontopontocerebellar system descends in the internal capsule and cerebral peduncle in close association with the pyramidal tracts, and it is interesting to note that both structures, the one subserving the static and the other the kinetic function of motility, receive their myelin sheaths subsequent to birth.

Both of the efferent systems of the cerebellum pass in the superior peduncles to special cellular divisions of the nucleus ruber (nucleus magnocellulatus and nucleus parvicellulatus), from which they descend as separate systems in the spinal cord, a paleorubrospinal system and a neorubrospinal system. I then conceive them as passing in separate systems to the sarcoplasm of the skeletal muscles.

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24. Tilney: Functional Significance and Principal Syndrome of the Cerebellum, Neurol. Bull. 2:289, 1919.

Before leaving the question of the central pathways, I would say a word in respect to a group of subordinate motor centers which are found scattered in the tegmental region of the brain stem, and usually referred to as prespinal centers of motility.

The prespinal or better supraspinal centers of motility represent a distinctly lower level of phylogenesis than the corpus striatum. They are intercalated in the pathway of the extrapyramidal system, of which they form a part and I believe contain representatives for both the kinetic and the static components of motility. With certain exceptions these cannot be differentiated in the present state of our knowledge. The red nucleus, however, may be cited as an example of a purely static center at the supraspinal level of motility. The manifestations of decerebrate rigidity are probably closely related to this supraspinal static mechanism.

MOTOR MANIFESTATIONS OF DISEASE

I would now pass to a consideration of the efferent system in its relation to symptomatology. Is it possible to recognize the existence of a static and kinetic system in the various motor manifestations of disease?

In the interpretation of such phenomena one should always consider the existence of positive and negative symptoms as differentiated by Hughlings Jackson.[25] The positive symptom occurs in lower centers from loss of the inhibitory action of higher centers. Many important motor disturbances are of this order and are referable to secondary functional discharge in lower centers.

In a general way, it may be said that a lesion of the kinetic system causes a disorder of movement, and a lesion of the static system, a disorder of tonus or of posture.

In many of the disturbances of motility both systems participate, although it is usually possible to indicate one or the other as the essential factor involved.

At the segmental level of the nervous system the tendon reflex is a typical example of kinetic function, as is the muscle tonus of static function. Langelaan [26] has recently shown that tonus consists of two components, a contractile tonus referable to the disk system and a plastic tonus of sarcoplasmic origin. These may also be recognized on direct percussion of the muscle by the quick contractions of myotatic irritability and the more persistent local reactions of the idio-muscular response.

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25. Jackson, Hughlings: The Evolution and Dissolution of the Nervous System, Brit. M. J. **1**:591, 1884.

26. Langelaan: On Muscle Tonus, Brain **38**:235, 1915.

A similar division of function is also demonstrable in reflex action, the kinetic system yielding reflex movements, and the static system reflex posture.

Such disorders of motility as paramyoclonus multiplex, myokymia, and fibrillary twitchings are spasmodic phenomena referable to the kinetic spinal mechanism, while myasthenia gravis is a paretic manifestation. The clonus of spastic paralysis and the tremor of paralysis agitans are both of kinetic origin. One is related to the neokinetic, and the other to the paleokinetic system. The more plastic tonicity which is also present in both of these forms of palsy is of sarcoplasmic origin and referable to the static system. Certain well marked differences in hypertonicity which characterize the two disorders I would refer to difference of static and kinetic representation in the skeletal muscle. Huntington's chorea and epilepsy are both of kinetic origin, the former is referable to the striatal and the latter to the cortical level of motility.

Other disorders of motility, referable to the corpus striatum, such as athetosis, dystonia, progressive lenticular degeneration and paralysis agitans, represent different degrees of involvement of its efferent and inhibitory systems. This phase of the subject has been considered in detail in my contributions to the symptomatology of the corpus striatum.[27]

Rarely, in epilepsy there are seizures which suggest a relation to the static system. These are characterized by unconsciousness and a sudden loss of postural control without convulsive manifestations (static epilepsy).

All forms of myotonia, cerebral, cerebellar, spinal and peripheral, would appear to be of sarcoplasmic origin and referable to the static system. There is also much in favor of the hypothesis that the tonic spasms of cerebellar origin and the tonic rigidity of tetanus are essentially contractile manifestations of sarcoplasm.

The nature of the Bárány reactions to labyrinthine stimulation also indicate, in my opinion, a relationship to static function and the sarcoplasm of muscle. I would recall the slow and quick components of labyrinthine nystagmus. The slow movement is primary and referable to excitation in the static sphere, while the quick movements are secondary, and of kinetic origin.

The relationship of the frontopontocerebellar system to various myotonic phenomena has already been emphasized by Kleist [28] and Kinnier Wilson. Mills [29] has also considered this question in a discus-

27. Hunt: The Efferent Pallidal System of the Corpus Striatum, a Consideration of Its Functions and Symptomatology, J. Nerv. & Ment. Dis. **46**:211, 1917.

28. Kleist: Ueber nachdauernde Muskelkontraktionen, J. f. Psych. u. Nerv. **10**:95, 1908.

29. Mills: Muscle Tonicity, Emotional Expression and the Cerebral Tonectic Apparatus, Neurol. Centralbl. **33**:1266, 1914.

sion of a cerebral tonectic mechanism. Kinnier Wilson and Walshe [30] have made the important observation that tumors of the frontal lobe anterior to the motor area may produce myotonia, a peculiar feature of which is the limitation of the disturbance to voluntary movements. Bumke [31] has also described a family disorder characterized by the occurrence of myotonia with both voluntary and automatic type of movement. It should be remarked that in all of these central forms of myotonia the usual electrical and mechanical evidences of myotonia congenita are absent, which proves the disorder to be of central origin.

These various clinical types of myotonia I regard as corroborative of the conception of a special system for the control of the higher function of static motility.

There is one organic symptom in particular which is closely related to the static function of the cerebellum, and that is the intention tremor. The intention or cerebellar tremor presents the characteristics of a loss of the static components of motility. The coarse tremor movements may be regarded as an effort of the kinetic system to compensate for the loss of the posturing or static functions of the sarcoplasm.

In a movement complex, the kinetic component governs the synergies of the movement itself, but is accompanied by the static function which guides, steadies and reinforces the movement in its course and fixes it in posture when the movement ceases. When this mechanism is disturbed the intention tremor results.

At a meeting of this Association five years ago, I [32] described Dyssynergia cerebellaris progressiva, an affection characterized by the gradual development of generalized intention tremors. On the basis of pathologic investigations not yet reported, I would refer this disorder to an atrophy of the efferent dentate system through which the cerebellum exercises control of the static system of motility (primary atrophy of the efferent dentate system).

In addition to these various somatic expressions of kinetic and static function, a similar division may be recognized in the mental sphere. Among these may be mentioned catalepsy, catatonia, and certain of the hyperkineses of psychic origin. A psychostatic and psychokinetic representation may therefore be postulated at the psychic level of motility.

This concludes my argument in favor of the two great systems of motility; it is only an outline, and were my time not limited it could be reinforced by many additional facts which would materially strengthen the hypothesis.

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30. Wilson and Walshe: The Phenomena of Tonic Innervation and Its Relation to Motor Apraxia, Brain **37**:119, 1914.

31 Bumke: Eine familiäre Form von Intentionskrampf Ztschr. f. d. Neurol. u. Psych. **4**:644, 1910.

32. Hunt: Dyssynergia Cerebellaris Progressiva, Brain **37**:247, 1914.

SUMMARY OF HYPOTHESIS

In conclusion, I would restate briefly the general principles underlying this conception of the efferent system. The function of motility, like sensation, is represented by special end organs and neural systems which subserve the various types and modalities of contractile function. Their phylogenesis is in strict accord with the evolutionary hypothesis. From the primitive movement and posture of the lower forms of life to the highest expressions of motility in man, the transition is a gradual one and is recorded in the efferent nervous system of man.

As the animal organism is subject to the mechanical laws of a physical world, two forms of movement, active and passive, have been evolved in the struggle for existence. The one is that passive contractility of the muscular system which maintains the organism in posture and equilibrium and counteracts the forces of gravity and atmospheric pressure. The other is a more active form of contractility by which the organism is enabled to move or change its position in space.

In some invertebrates these two forms of motility are readily distinguished and are subserved by separate muscles. In higher forms these two important functions of muscle tissue are united in the muscle fiber, but are controlled by separate neural systems.

It is therefore possible to trace the evolution of the static and kinetic systems of motility from the lowest to the highest levels of the neural mechanism.

ACQUIRED DOUBLE ATHETOSIS*

(DYSTONIA LENTICULARIS)

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In 1916 I[1] reported several cases of a disorder occurring in one family, which seemed to me to belong to the group known as pseudosclerosis, probably a family form of a lesion of the lenticular nucleus, and in that paper I grouped the various disorders attributed to disease of the lenticular nucleus. These were the pseudosclerosis of Westphal and Strümpell, Huntington's chorea, Parkinson's disease, spastic pseudobulbar palsy with contractures and choreo-athetoid movemeünts of Oppenheim and Vogt, and Freund and Vogt, Oppenheim's dystonia musculorum deformans, and double athetosis. To this list I added von Bechterew's hemitonia apoplectica and certain forms of carbon monoxid poisoning, and I am inclined to add a certain form of paramyoclonus multiplex resulting from lethargic encephalitis. To the list might also be added arteriosclerotic muscular rigidity and certain forms of senile dementia, which Strümpell thinks belong to his amyostatic syndrome. He refers to a paper on the arteriosclerotic form of muscular rigidity by O. Förster, but is unwilling to attribute to arteriosclerosis of the brain arteries so great causal and primary significance as Förster has done. He thinks other etiologic factors are to be considered and that while arteriosclerosis is a frequent accompanying condition, it is not a conditio sine qua non.

Nonne says that numerous findings in lethargic encephalitis show that acute inflammation of the basal ganglions may cause the amyostatic syndrome. It seems to me that the involuntary jerkings of lethargic encephalitis probably have their origin in these structures, whether they assume a choreiform or myoclonic type. A few months ago a case of the myoclonic form of lethargic encephalitis with intense muscular contractions of the myoclonic type in various parts of the body occurred in my service and terminated fatally. I found in microscopic sections a pronounced cellular infiltration of the region of the basal ganglions, but not confined to them. It occurred diffusely in the basal part of the temporal lobe. The intense perivascular

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* Read before the Forty-Sixth Annual Meeting of the American Neurological Association, New York, June, 1920.

1. Spiller, W. G.: Pseudosclerosis, J. Nerv. & Ment. Dis. **43**:23 (Jan.) 1916.

cellular infiltration of the region of the aqueduct of Sylvius, so common in the usual form of lethargic encephalitis, did not occur in this case.

REPORT OF A CASE

History.—M. A., a girl, aged 16, was admitted to the Philadelphia General Hospital, Sept. 5, 1882, and died there Dec. 24, 1919. The notes taken when she was admitted stated that her father was dead, but her mother, one brother and one sister were living and well. The patient's intelligence was said to be good. When she was about 5 years of age it was noticed that she had begun to drag her left lower limb and that her left upper limb was useless. Other parts of the body became successively affected and the paralysis assumed a spastic character, the head became drawn to the left, the body arched to the left, the knees flexed and the feet placed in the position of talipes varus. Marked incoordinate movements were observed in the hands and feet; the limbs on the left side became useless, but the right hand still had some voluntary function. She could not walk and could not sit in a chair unless tied in it. Articulation was difficult and defective.

Dr. George Wilson made some notes on Jan. 29, 1915. She had been in the Philadelphia General Hospital thirty-three years. She was irritable and had an exceedingly violent temper. She objected strenuously to being exam-

Fig. 1.—Spastic contractions with athetoid movements. These photographs were taken by Dr. Ralph Pemberton some years before the death of the patient.

ined and on one occasion called the examiner an abusive name with unusual distinctness. She had a peculiar explosive speech, which was indistinct; occasionally words were said distinctly. One who associated with her constantly was able to understand what she said. She had difficulty in chewing and swallowing and had to be fed.

Examination.—She appeared shrunken. Her irides seemed to react to light and in convergence. She had no nystagmus and no exophthalmos. Her lower jaw was frequently drawn downward and to the left and the movements were typically athetoid. Her upper limbs had little voluntary power. The fingers were tightly clasped into the palms and the forearms were supinated or pronated slowly and unevenly. The upper limbs were hypertonic and the tendon reflexes could not be obtained satisfactorily.

The lower limbs were extremely spastic and presented athetoid movements, the right foot was strongly inverted and the right great toe extended in the movements, while the left foot was placed so strongly in the position of

equinus that the toes were more posterior than the heel. Plantar stimulation gave no response except athetoid movements, which seemed more pronounced when external stimulus was applied to the feet. The patellar reflexes were not obtained on account of the spasticity. At times when the athetoid movements relaxed the entire right lower limb seemed to go into clonus. The sensation of pain was preserved.

On Feb. 21, 1916, the condition was about the same. All the limbs showed contractures and there was some muscular atrophy. Mental deterioration was marked.

On June 10, 1916, it was noted that she agreeably cooperated in the examination to the best of her ability. While mentality was poor she showed no attacks of exaltation or depression, and no errors of sense perception or of judgment.

Her muscular system was continually in a universal athetosis. The arms, hands, legs and feet were most involved and to an extreme degree, the neck was less involved, and the trunk only slightly involved. There was a spastic scoliosis. The tendon reflexes were not obtained, possibly because of the spasticity. The muscles were moderately atrophied. Both pupils showed myosis. On Dec. 28, 1916, she had periodic attacks of diarrhea. She had myocarditis and her death on Dec. 24, 1919, was attributed to this cause.

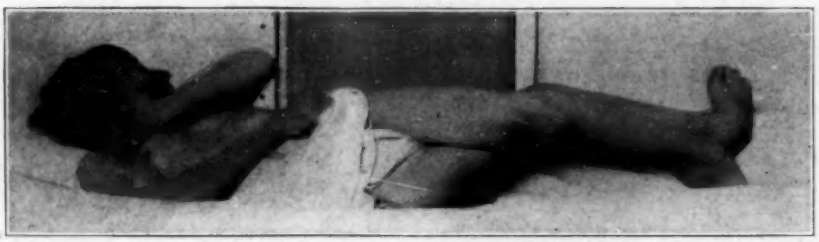

Fig. 2.—Spastic contractions with athetoid movements.

Necropsy Examination.—The necropsy examination was performed by Dr. Edward Weiss. The liver showed passive congestion; its weight was 510 gm. It was long and narrow, its surface was granular, the capsule was transparent and the substance finely mottled. It showed marked congestion of the central acini of the lobules with dilatation of the contributing sinusoids and atrophy and pigmentation of the surrounding liver cells, with slight increase in the supporting tissue. The diagnosis was chronic passive congestion and red atrophy.

The spleen showed passive congestion. It was small and firm; the interior was dark red; the trabeculae were well marked. It weighed 70 gm. It showed hyperplasia of the follicles, considerable increase in connective tissue supportive structure and splenic capsulitis. The diagnosis was chronic diffuse splenitis.

The left kidney weighed 80 gm.; the right 90 gm. They showed slight chronic interstitial nephritis. The left kidney was small and firm and cut with increased resistance; the capsule stripped with difficulty, leaving a finely

granular surface; the cortex was narrow and the cortical striations distinct. The left suprarenal gland grossly was normal. The right kidney and suprarenal gland were about the same as the left.

The length of the body was 50 inches. The brain and cord weighed 1,170 gm. They were studied by me. The convolutions of the frontal lobe were distinctly smaller than those of the rest of the cerebrum, but there were no sclerotic areas in the cortex.

Microscopic Examination.—A horizontal section was made through the upper part of the basal ganglions. Each lenticular nucleus was about one-half the normal size. The globus pallidus on each side was firm but the putamen on each side had a worm eaten appearance and contained numerous small holes. A second horizontal section was made through the cerebrum about 1 cm. below the first section, and the basal ganglions from the left cerebral hemisphere in this upper piece were cut in serial sections. The basal ganglions on the right side were cut in serial section below the second cut. As the basal ganglions from the two sides had the same macroscopic and microscopic appearance, it was not considered necessary to cut all parts of them, especially as it was desirable to keep a portion of them intact on account of their gross appearance.

Sections from the tip of the right frontal lobe were stained with thionin to determine the cellular condition. They did not show any distinct pathologic condition. The Betz cells of the paracentral lobules were greatly altered, the nucleus was eccentric and the chromatolysis was pronounced, but the smaller nerve cells of the paracentral cortex did not appear to be greatly altered. Weigert hematoxylin sections of the paracentral lobules were normal.

The nerve cells of the left island of Reil were in good condition. Nowhere could an increase in size of the neuroglia nuclei be found. The left putamen was made up of shreds of tissue containing numerous neuroglia nuclei but only here and there a nerve cell, small and much altered, was found. The Weigert hematoxylin stain showed numerous separate, wavy, medullated fibers in the putamen, each fiber standing out distinctly on the brown background. The blood vessels of the putamen were much thickened and the perivascular spaces were much enlarged.

The globus pallidus was atrophic, as was also the nucleus caudatus, but neither showed the peculiar tissue seen in the putamen. The external and extreme capsules were well defined by the Weigert hematoxylin stain. The internal capsule, both the anterior and posterior limbs, was normal. The thalamus was about normal in size and appearance.

The putamen of the right side of the brain showed the same condition as that of the left side. The perivascular spaces within it were greatly enlarged. The globus pallidus, aside from a diminution in the number of medullated nerve fibers and a smaller size, was not greatly altered. The ansa lenticularis was probably a little smaller than normal. The cells of the nucleus ruber were in good condition and the nucleus was of normal size. The corpus dentatum appeared to be normal, but the cells were much pigmented. The anterior cerebellar peduncles were normal.

The nerve cells in the anterior horns of the spinal cord in the cervical and lumbar regions by the thionin stain appeared on the whole to be normal, except that here and there an intensely altered cell was found, in which the cell body was swollen, the nucleus was displaced and chromatolysis was pronounced. The pyramidal tract was normal throughout the brain and spinal cord.

DIFFERENTIAL DIAGNOSIS

Lenticular Degeneration.—When one reads the clinical description Wilson [2] gives of his progressive lenticular degeneration, the resemblance to bilateral acquired athetosis is striking. He says: It consists of involuntary movements, nearly always a bilateral tremor of both upper and lower extremities, the head and trunk also being sometimes involved—a tremor usually rhythmical but occasionally irregular, and increasing with volitional movement; there is pronounced spasticity of the limbs and of the face, the latter being usually set in a spastic smile, while in the later stages contracture of the

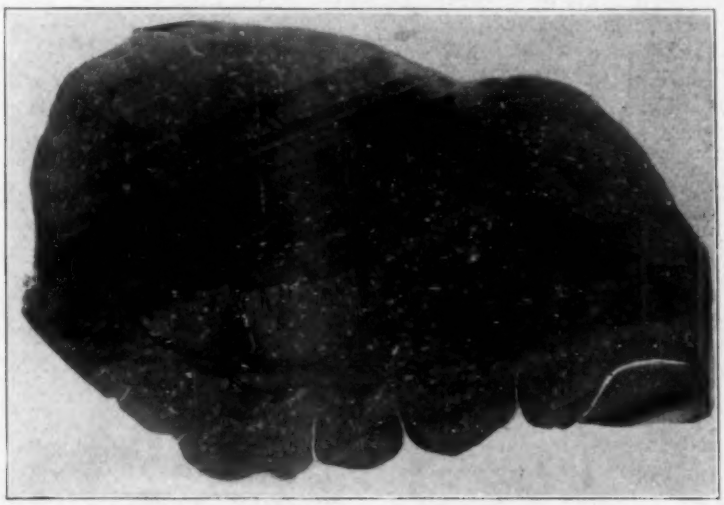

Fig. 3.—Normal section through the basal ganglions. Compare this with the section represented in Figure 4 taken at about the same level from the case of double athetosis.

limbs develops; there is dysphagia and dysarthria, the latter eventually degenerating into complete anarthria; there is sometimes spasmodic laughing and emotionalism. As a result of the extraordinary degree of stiffness of the musculature there is considerable difficulty in maintaining equilibrium. Little or no true paresis or paralysis occurs, however, as most ordinary movements, if not all, can be executed, though it may be slowly and feebly, and there is some weakness. In some cases certain mental symptoms, of a transitory nature, manifest themselves. There is no sensory disturbance and no alteration in the cutaneous reflexes. If the abdominal reflexes are absent (apart from muscular rigidity) or the plantary reflex is of

2. Wilson: Brain **34**:295, 1912.

the extensor type, the syndrome is no longer pure. This, Wilson holds, is the clinical picture in pure, uncomplicated, bilateral lesions of the lenticular nucleus, and more generally of the corpus striatum, provided they are of sufficient size and of adequate duration.

Wilson says Gowers, Ormerod, Homen and he (Wilson) have been struck by the odd way in which the patients, seemingly so inarticulate, have occasionally been able to utter words or phrases with comparative ease and distinctness. This was especially observed in my case of double athetosis when, on one occasion, the patient said "damned fool" with great distinctness, whereas usually she could hardly be understood.

The lesions Wilson found in the lenticular nucleus in his cases consist of glial overgrowth which disintegrates and breaks down; there is often an immense increase in glial nuclei; the nerve fibers and nerve cells of the normal nucleus disappear; "Körnchenzellen" and macrophages are frequently present in numbers. Even when the cavity formation is extreme there are no signs of obliterative endarteritis in the blood vessels, the perforating lenticulostriate vessels and their branches. In advanced cases there are degeneration of the ansa lenticularis, relative atrophy of the corpus Luysii, partial degeneration of the lenticular bundle of Forel and of the strio-Luysian fibers and degeneration of striothalamic fibers. Apart from the degeneration in the lenticular nucleus, the changes in the brain are insignificant. Thus, the pathologic finding is bilateral symmetrical degeneration of the putamen and globus pallidus, especially of the former. The caudate nucleus is often somewhat atrophic, but never to the same extent. The Betz cells are intact and the cerebral cortex offers little definite alteration.

Although Wilson found no important cortical changes in his cases, others have reported such findings. It is impossible to explain all the symptoms of progressive lenticular degeneration by lesions of the lenticular nucleus, and this has impressed Strümpell, who states that the psychic symptoms (dementia, excitement and other symptoms) cannot be attributed to the disease of the lenticular nucleus.

In the case of progressive lenticular degeneration reported by Lhermitte [3] the findings in the lenticular nucleus closely resembled those in my case of athetosis. He found atrophy of the putamen and caudate nucleus, and the putamen contained lacunes of disintegration. The vessels were normal in these lesions. The picture he gives of the lesions resembles the findings of my case but the putamen appears less intensely affected. He says the nerve cells of all regions of the cerebral cortex were atrophied and full of lipochrome granules.

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3. Lhermitte: Semaine méd. **32**:121 (March 13) 1912.

Schütte [4] reported a case which he thought resembled progressive lenticular degeneration, and he found decided alteration of the brain, especially of the frontal lobe, where the nerve elements were destroyed in places. The remaining cerebral cortex was diffusely affected but much less intensely. The basal ganglions showed some change in the ganglion cells and increase of neuroglia, but only in slight degree, and yet the symptoms were not essentially different from those of Wilson's cases. Schütte questions whether the lenticular nucleus is the seat of the disease. He also found the liver diseased.

In the case of progressive lenticular degeneration reported by Pfeiffer,[5] in addition to lenticular lesions the cerebral cortex was much altered. The ganglion cells in the frontal region were more

Fig. 4.—Basal ganglions from the case of double athetosis. Compare the lenticular nucleus with the lenticular nucleus in Figure 3. The section is from the left cerebral hemisphere.

severely affected than in other parts of the cortex. A swelling of the cells was observed, but shrinkage and other types of cellular disease were likewise frequently encountered, limited to the small and medium sized cells. In the motor region the giant cells were not perceptibly affected, but the commissural and associated neurons were altered.

In Pollock's [6] case of progressive lenticular degeneration, in addition to lesions of the lenticular nuclei some cortical changes were found but the Betz cells seem to have been little altered.

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4. Schütte: Arch. f. Psychiat. **51**:334, 1913.
5. Pfeiffer: J. Nerv. & Ment. Dis. **45**:289 (April) 1917.
6. Pollock: J. Nerv. & Ment. Dis. **46**:401 (Dec.) 1917.

Pseudosclerosis.—The pseudosclerosis of Westphal and Strümpell apparently is generally regarded by German writers as identical or at least closely allied with Wilson's progressive lenticular degeneration, and some go so far as to refer to both conditions collectively as the Westphal-Strümpell-Wilson syndrome (Thomalla). Wilson recognized the resemblance between the two disorders in an appendix to his paper in *Brain* and in Lewandowsky's "Handbuch," and Strümpell has expressed the opinion that they are the same disease.

Otto Maas [7] came to the conclusion that no distinction could be made clinically or pathologically between the two disorders. Thomalla [8] says that in all cases of pseudosclerosis carefully studied histologically, changes were found in the lenticular nucleus, in other ganglions of the cerebrum and often in the nucleus dentatus. He asserts that von Maas and von Dziembowski have demonstrated the identity of the two diseases. I have not been able to obtain the paper by the latter author, but Thomalla referring to it says that von Dziembowski in his first case found no changes in the lenticular nucleus and attributed all the symptoms to disease of the liver and alteration of the blood; nothing is said about the method of the histologic examination.

It is therefore worth while to study some of the recent papers on pseudosclerosis. This is a term used chiefly by German writers.

Hösslin and Alzheimer,[9] in a case of pseudosclerosis, found the convolutions in general small and the sulci widened. Nowhere was cellular infiltration found. The glia of the brain was altered everywhere and there were many large glia cells. The entire central nervous system was affected, especially the corpus striatum, thalamus, regio subthalamica, pons and nucleus dentatus. The cells after alcohol hardening were stained with toluidin blue.

A. Westphal [10] in his case of this disease found alteration of the glia nuclei, especially in the basal ganglions and the nucleus dentatus of the cerebellum, corresponding with those described by Alzheimer, but less pronounced and indicating an earlier stage of the disease.

Otto Mass' case of the same disease showed bilateral degeneration of the lenticular nucleus and nothing pathologic in the optic thalamus and caudate nucleus. In the lenticular nucleus the nerve cells had almost entirely disappeared, but nerve fibers coming from outside the lenticular nucleus and entering it were preserved. The neuroglia of the lenticular nucleus showed proliferation and many astrocytes.

7. Maas: Neurol. Centralbl., 1918, No. 1, p. 16.
8. Thomalla: Ztschr. f. d. ges. Neurol. u. Psychiat. **41**:311, 1918.
9. Hösslin and Alzheimer: Ztschr. f. d. ges. Neurol. u. Psychiat. **8**:183, 1912.
10. Westphal, A.: Arch. f. Psychiat. **51**:1, 1913.

Maas says changes have been found in pseudosclerosis not only in the liver, but in the spleen and other glands. Fleischer, in reporting a case of pseudosclerosis, is said to have been the first to emphasize that liver changes belong to pseudosclerosis. Völsch later also pointed out the importance of the liver findings. Rausch and Schilder [11] say the liver changes in pseudosclerosis are identical with those in Wilson's progressive lenticular degeneration. The brain changes are more diffuse in pseudosclerosis, but Stöcker shows that this is not always true as he found changes in the entire brain in Wilson's type.

Rumpel [12] believed the liver which he studied from a case of pseudosclerosis was the result of fetal developmental disturbance, probably from congenital syphilis.

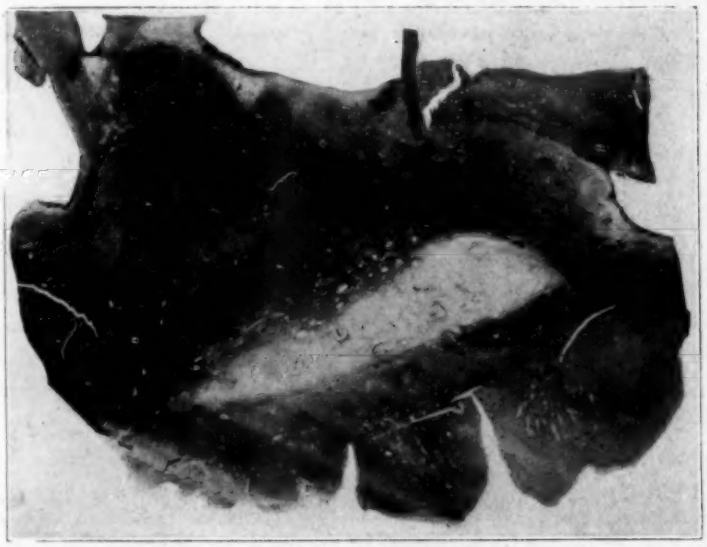

Fig. 5.—Section through the right basal ganglions from the case of double athetosis. The degeneration affects the putamen and the tissue consists of loose neuroglia. There is no large cavity in the putamen. It has been difficult to photograph these sections.

Athetosis and Lenticular Degeneration.—Are the changes of the nerve cells of the cerebral cortex independent of the alteration of the lenticular nucleus or are they secondary to it? It has been generally believed that no important connection exists between the ceberal cortex and the lenticular nucleus, and although this view has been difficult to accept, anatomic investigations have seemed to establish it. Wilson holds that the direct connection of the corpus

11. Rausch and Schilder: Deutsch. Ztschr. f. Nervenh. **52**:414, 1914.
12. Rumpel; Deutsch. Ztschr. f. Nervenh. **49**:54, 1913.

striatum with the cerebral cortex is minimal. Recently von Economo,[13] however, has thrown much light on this question. He believes he has demonstrated an anatomic connection between the lenticular nucleus and certain parts of the cerebral cortex, a striotemporal and a strioparietal connection. This, he says, is contrary to the teaching of Déjerine, who denied connection from the cortex to the corpus striatum, and said that only collaterals from the pyramidal tract fibers entered the corpus striatum, as no cortical lesions caused atrophy of the corpus striatum, such as is seen in the optic thalamus. Von Economo holds that as there are association tracts between different parts of the cerebrum, and a lesion of one part does not cause atrophy of other parts connected with it by these association tracts, so there exists a connection between the corpus striatum and the cerebral cortex, and the tracts discovered by him, the striotemporal and strioparietal, are examples of such an association. It would be strange, he thinks, if the corpus striatum were isolated from the rest of the cerebrum except by its striofugal connections with the optic thalamus, corpus Luysii and nucleus ruber. Von Economo regards the tonic spasm without reflex exaggeration as a pathognomonic symptom of disease of the lenticular nucleus.

It is possible, therefore, that the alteration of the cells of the cerebral cortex has a close connection with the alteration of the lenticular nucleus. The changes in the Betz cells in my case of athetosis, evidently far greater than any alteration of the cortex observed by Wilson in his cases, may have some connection with the greater severity of involuntary movement in my case, in which there was athetosis of an extreme type in comparison with the tremor of Wilson's cases; and yet Madame Vogt did not find intense change in the cortical nerve cells in her studies.

The most striking difference in the description given by Wilson of progressive lenticular degeneration and one of acquired athetosis is in relation to the duration of the disorder and the presence of tremor, as in bilaterial athetosis the involuntary movement is not regular and is unlike that described by Wilson; and yet in Gowers' case which Wilson accepts as belonging to his type, it is stated that the fingers at times were extended and slowly moved in the irregular way characteristic of athetosis. Wilson states that tremor and athetosis may occur together, but they are sufficiently distinct to be described apart. In another place he distinctly states that associated movements and athetoid movements do not occur in progressive lenticular degeneration.

It remains to be proved, I think, whether the involuntary movements in progressive lenticular degeneration are practically always of

13. Von Economo: Ztschr. f. d. ges. Neurol. u. Psychiat. **43**:173, 1918.

the tremor type and are characteristic of lesions confined to the lenticular nucleus. My case of athetosis is proof to the contrary, so is Fischer's [14] case of athetosis and Thomalla's case of torsion spasm; especially so are those studied by Madame Vogt in association with others. Von Economo has shown by a case of his own and by others that in progressive lenticular degeneration spontaneous movements may be entirely absent. Lenticular lesions may therefore cause involuntary movements of different types.

Wilson states, in comparing his cases with those of Madame Vogt,[15] that in his cases there were no associated movements or any type of

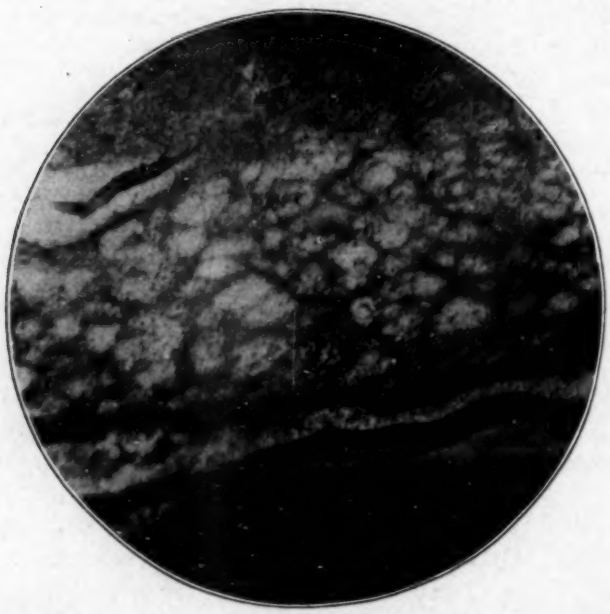

Fig. 6.—Section under greater magnification through the putamen, showing the loose neuroglia tissue. A blood vessel with an enlarged perivascular space is seen passing entirely across the section. (Photographs of the brain are by Dr. A. J. Smith.)

athetosis. These differences he regards as important, for progressive lenticular degeneration is a disease *sui generis,* and is not to be considered as a variety of "athétose double." Madame Vogt believes that the syndrome of the corpus striatum is an "athétose double pure," but Wilson is more inclined to associate tremor than athetosis with the syndrome.

14. Fischer: Ztschr. f. d. ges. Neurol. u. Psychiat. **7**:463, 1911.

15. Oppenheim and Vogt: J. f. Psychol. u. Neurol. **18**: 1912. Freund and Vogt: Ibid.

Wilson points out that in Anton's case, which he (Wilson) associates with cases of athetosis, the involuntary movements in the lower limbs were more like tremor. There was therefore in this case an association of tremor with lenticular disease.

Madame Vogt's findings are: In the case of infantile pseudobulbar paralysis with athetosis reported by her with Oppenheim, the caudate nucleus and putamen were much affected. The globus pallidus was atrophied in its dorsal portion. The ansa lenticularis appeared little altered. Nothing abnormal was found in the cerebellum or in the cerebral cortex. The internal capsule appeared to be unusually large on account of the change in the corpus striatum. Madame Vogt thought the parts destroyed must have had a regulating and inhibiting effect on the motor system.

In the case reported by her with Freund, the cerebral cortex showed no noteworthy alteration. Here also was found only atrophy and état marbré in a part of the caudate nucleus and the putamen. The rest of the brain was intact; even the Betz cells were normal.

Cécile Vogt, depending on the case of Anton, the cases she studied with Oppenheim and Freund and the two brains from Barré, believes the état marbré and atrophy of the corpus striatum produce the syndrome of the corpus striatum, namely, double athetosis without or almost without paralysis and without intellectual deficit or without pronounced intellectual deficit.

Strümpell [16] reports two clinical cases in brother and sister which he does not fully identify with Wilson's type, but brings into close relationship with it. Rigidity was the most characteristic feature in his cases and tremor was so slight and fine that without careful observation it would have been overlooked, and it was not always present. In one of these cases athetosis was especially marked in the hands and less so in the feet; in the other case athetosis was not observed in the hands, but it was pronounced and constant in the toes though not intense. These cases show the relation between progressive lenticular degeneration and bilateral athetosis.

Anton's [17] case was one of chorea with athetosis. He found almost symmetrical isolated lesions of the posterior half of the putamen of the lenticular nucleus. The optic thalamus and internal capsule were normal. He attributed the lesions of the putamen to thrombosis of the several small vessels supplying the putamen and passing through the substantia perforata. Such thrombosis might be caused in children by cachexia and fever. He quotes Kolisko as showing that the

16. Strümpell: Deutsch. Ztschr. f. Nervenh. **54**:207, 1915-1916.
17. Anton: Jahrb. f. Psychiat. **14**:141, 1896.

arteries supplying the caudate nucleus and the globus pallidus come at a sharp angle from the anterior cerebral artery, and the current is opposite to that in this artery.

Oskar Fischer mentions that although lesions in a location typical for hemichorea are frequent, hemichorea seldom occurs. Why in one case chorea occurs and in another is wanting he cannot explain. The same kind of lesion in the same location in one case may cause hemichorea, in another hemiathetosis or hemitremor. There is no satisfactory explanation for this.

He reports a case of double athetosis of gradual development in which he found granular pigmentary degeneration of the ganglion cells in the globus pallidus of each lenticular nucleus. The boy was normal until 14 or 15 years of age, but between 15 and 17 the athetosis probably developed. The necropsy showed great diminution in the size of the globus pallidus, it appeared shrunken and was of brownish color, and was more intensely colored in its anterior portion, but showed no softening or hemorrhage. The atrophy of the lenticular nucleus was evident in Weigert sections. The cerebral hemispheres were smaller than normal, and the white matter less developed, but this was much less than the atrophy of the lenticular nucleus, although the atrophy was not equally intense in all parts of the nucleus. The putamen was distinctly atrophied but not nearly so much as the globus pallidus, which was about half the normal size. The globus pallidus was poor in medullated fibers, more so in the anterior portion, which was of much the same color as the putamen. The optic thalamus and internal capsule were distinctly smaller but the caudate nucleus showed no change.

The pallor of the globus pallidus was caused by a scarcity of medullated fibers and the small size of the medullated fibers preserved. The ganglion cells of this structure were greatly changed; in Nissl stain they were enlarged and rounded, and the nucleus was swollen and the nucleolus enlarged. The cell body contained numerous fine granules and other changes were apparent. The globus pallidus alone showed pathologic change. No changes were found in the anterior cerebellar peduncles.

LOCATION OF LESIONS IN ATHETOSIS

Schilder [18] shows that in athetosis the lesion was found in different locations, and he classifies the cases as follows:

Lenticular Lesions:

1. Anton's case in which pathologic changes were found only in the putamen of each lenticular nucleus and the left ansa lenticularis was small.

18. Schilder: Ztschr. f. d. ges. Neurol. u. Psychiat. **7**:219, 1911.

2. Berger's case in which athetosis was confined to the right side. A cavity was found in the left lenticular nucleus.

3. Oppenheim and Vogt's case in which there was pseudobulbar palsy with athetosis and changes in the corpus striatum.

Optic Thalamus Lesions:

1. Herz's case, in which there was athetosis on the right side. A cyst was found in the optic thalamus; the nucleus ruber, anterior cerebellar peduncle and nucleus dentatus were altered.

2. Muratow's case of athetosis. A gumma was found in the optic thalamus and the anterior cerebellar peduncle also showed a lesion.

Lesions were found in the nucleus ruber in the cases of Halban and Infeld, and Marie and Guillain.

The anterior cerebellar peduncle was affected in Bonhöffer's case.

A tubercle was found in the cerebellar hemisphere in Pineles' case.

In a case of unilateral athetosis reported by Schilder a tumor was found in the lenticular nucleus. Another lesion was found in the cerebellum.

Nonne has recently expressed the opinion there can no longer be any doubt that lesions of the optic thalamus and corpus striatum are responsible for athetosis and certain forms of chorea and paralysis agitans.

DYSTONIA MUSCULORUM PROGRESSIVA IN CORPUS STRIATUM GROUP

It is interesting to see that dystonia musculorum deformans has a pathology placing the disorder in the group of corpus striatum diseases.

In a case of this kind reported by Curt Thomalla there was intense alteration of the putamen of the lenticular nucleus. The putamen on each side was abnormally small; its tissue was below the level of the surrounding structures in a section through the brain; it was of softer consistency and was fissured (zerklüftet). The putamen showed softening, its parenchyma was completely destroyed, ganglion cells and nerve fibers had almost entirely disappeared, and the neuroglia had only partially proliferated to take its place. A spongy porous glia structure replaced the normal tissue of the putamen, in which the glia nuclei were much multiplied, and numerous fatty granular cells were in its meshes. Astrocytes were few. The vessels were conspicuous in this gliar reticulum, but were not quantitatively increased. A feeble round cell infiltration was found in the outer coat of certain vessels. The pathologic condition was sharply defined from the cortex of the island of Reil.

The nucleus caudatus showed no noteworthy change, and the bands of tissue connecting the nucleus caudatus and the putamen through

the anterior limb of the internal capsule were not altered. The globus pallidus was not appreciably altered and the cortex of the island of Reil was intact.

The liver was in a condition like that described by Wilson in his cases and in cases of pseudosclerosis. The inner structure was different and was believed to indicate a defect of development. The pathologic findings were reported only in a preliminary manner.

Thomalla's patient showed transitory but typical athetoid movements, and he refers to the fact that double athetosis has often been mentioned in association with torsion spasm. His case of torsion spasm is the first in which there has been a necropsy examination. He says the alteration of the lenticular nucleus was almost identical with that in Wilson's cases, as was also the alteration of the liver.

He quotes Oulmont as saying double athetosis is a primary bilateral hemiathetosis, almost always beginning in the first years of life, usually with idiocy. Lewandowsky believed it to be more than bilateral hemiathetosis, but on account of deficient power of dissociation it is a form of generalized but not identical associated movements. There is a rhythmical character and slowness of the athetoid movements, spasmus mobilis, strongly influenced by emotion, involvement of the face, lack of dissociated movements in the face, diplegia, disturbance of reflexes and a positive Babinski reflex. Often there are contractures. The irritative motor phenomena may persist when the patient is quiet, even in sleep. Imbecility is almost constant, but many authors have observed that the intelligence is intact.

Thomalla speaks of the clinical resemblance of his case of torsion spasm to double athetosis and says Ziehen and Schwalbe also observed the similarity between their cases and athetosis. Oppenheim also accepted a relationship between these two disorders, and spoke of transitional cases. The relationship has been recognized also by Flatau, Sterling and others.

Thomalla points out that the Babinski reflex was absent in Klempner's case of double athetosis. He regards his case as a transitional one between torsion spasm and double athetosis, as he does also Flatau's and Fischer's cases. He says the liver in A. Westphal's case of pseudosclerosis was like the liver in his own case. He calls attention to the resemblance between torsion spasm and Wilson's progressive lenticular degeneration in the muscular rigidity of the entire body, including the muscles of mastication and speech; in the predominance of the flexors with gradually developing flexor contracture, etc. The resemblance between double athetosis and Wilson's type is shown by the muscular rigidity, the gradual development of contracture, the dysarthric and dysphagic disturbances, progressive character, family occurrence and psychic disturbances.

He discusses the resemblance between bilateral athetosis, pseudosclerosis, progressive lenticular degeneration and torsion spasm. In all there is disturbance of tonus shown by: spasms in double athetosis and torsion spasm; muscular rigidity in Wilson's type and pseudosclerosis, differing in all these forms from spasms of pyramidal tract origin; involuntary movements, disturbances of speech and swallowing in all, gradually developing contracture of the limbs in all, even to complete rigidity, masklike face and boardhard abdominal muscles. In all there is disease of the lenticular nucleus. The cases of double athetosis with pyramidal tract involvement must be separated from the group. Thomalla proposes for the whole group the name of dystonia lenticularis. He says as yet similar findings in the liver of patients with double athetosis do not exist; perhaps they have been overlooked; perhaps they are really lacking. In all the groups there is absence of atrophy, paresis, electrical alteration or pyramidal tract symptoms. In many cases cerebral cortical changes have been found.

J. Ramsay Hunt[20] found as the cause of juvenile and early adult paralysis agitans progressive atrophy of the globus pallidus, consisting of atrophy and diminution in number of the motor cells of the globus pallidus, the basal ganglion of Meynert, and especially the cells of similar type scattered through the caudate nucleus and the putamen (neostriatum). The evidences of cellular atrophy were especially well marked in the large cells of the neostriatum. There was a corresponding increase of the glia nuclei and a thinning of the fibers of the ansa system—striohypothalamic radiations. The small ganglion cells of the neostriatum were intact and the pyramidal tracts showed no signs of atrophy or degeneration. Hunt found no essential lesions except of the corpus striatum in juvenile paralysis agitans.

CAUSE OF INVOLUNTARY MOVEMENTS

Strümpell explains tremor and athetosis as the result of faulty innervation of the muscles which fix a limb in any position, when the agonistic and antagonistic muscles are not simultaneously innervated to the normal degree. If the innervation of one group occurs at a different time from that of the other, involuntary movement results; tremor if the alternate innervation is regular; athetosis if it is irregular. The proper innervation of the agonistic and antagonistic muscles of a part which permits proper fixation of the part is called by Strümpell myostasia, whereas the disturbance of this innervation so that steady fixation is impossible, is called the amyostatic syndrome or myastasia. Paralysis, ataxia, spasm, which are disturbances of muscle movement, he calls in distinction, the myodynamic complex or the pyramidal tract

20. Hunt: Brain **40**:58, 1917.

syndrome. In the type described by Wilson the amyostatic syndrome is especially marked, as shown by tremor, muscular rigidity, rigidity of expression, and other symptoms.

Strümpell says that intestinal symptoms, dyspepsia, diarrhea and abdominal pain have been observed only in the pseudosclerosis or the type described by Wilson. This discussion by Strümpell is interesting, but giving all the types the name of amyostatic syndrome does not explain why they occur. This question has puzzled all students of the subject. Gowers attributed involuntary movements to lesions of the basal ganglions. Von Monakow does not believe tremor is caused by direct irritation of the pyramidal tract. He believes lesions of the corpus striatum cause irritation of the motor cortex and that from this source arise the impulses for choreiform movement. Wilson attributed involuntary movements to extrapyramidal fibers, such as are (1) in the tract from the nucleus dentatus through the superior cerebellar peduncle, to the nucleus ruber of the opposite side and from this to the outer part of the thalamus opticus and to the sensory and motor cortex. Involvement of this tract must cause athetoid and choreiform movements.

(2) In the tract arising in the nucleus lenticularis, through the ansa lenticularis in the regio subthalamica, the corpus Luysii and the nucleus ruber, from here as von Monakow's tractus rubrospinalis to the cells of the anterior horn of the spinal cord.

The movements are supposed to occur because of loss of corticopetal impulses which should pass to the cortex through the cerebello-rubro-thalamo-cortical tract. Lesion of this tract anywhere will cause posthemiplegic chorea by preventing the inhibitory impulses from reaching the cortex. This view is confirmed by the statement of deVries that extirpation of motor cortical areas in those with hemiparesis and choreo-athetoid movements causes cessation of these movements. Wilson ascribes the tremor to a destructive lesion of the lenticulo-rubro-spinal system. On account of the loss of this secondary motor tract the normal inhibiting impulse which the corpus striatum exercises over the anterior horn cells of the spinal cord are lost. The uniform innervation of the anterior horn cells is thus made impossible and stronger impulses pass to them by the pyramidal tract. Every voluntary movement increases the tremor, and this is usually true of the athetoid and choreiform movements.

HERNIATION OF THE BRAIN*

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The falx and tentorium constitute an important protection against any sudden impacts of pressure by keeping apart heavy portions of the brain, but they also provide an opportunity for trouble in case of swelling or need of displacement.

From the material that has gone through the Neurological Laboratory of the Henry Phipps Psychiatric Clinic, I have selected some cases illustrating various types of herniation under the falx and under the tentorium and into the foramen magnum. They give an interesting quasi-experimental picture of the topography and at least in two instances collateral consequences of possible importance among the so-called distant symptoms of brain tumor.

The line along which the tentorium can produce depressions passes over the uncus along the subiculum of the cornu ammonis.

The case A. M., with glioma of the left opercular region, shows, beside a moderate cerebellar wedging into the foramen magnum, a characteristic sagittal "tentorial line" on the left uncus, not to be taken for a sulcus semi-annularis Retzius (Figs. 1 and 2).

The case D. W., with a glioma of the anterior and middle hindbrain segment, shows similar tentorial lines due to slight hydrocephalus (Figs. 3 and 4).

A most extensive prolapsing glioma of the left temporal lobe (G. McL.) with death in coma, shows a frontal herniation under the falx and a marked subtentorial herniation of the uncus (Fig. 5).

In M. B. (Figs. 6 to 8) a cancer metastasis with a cyst in front of the paracentral lobule leads to herniation of the fornicate gyrus under the falx and subtentorial herniation of part of the uncus and of both subicula, with a striking impingement on the calcarine branch of the posterior cerebral artery and collapse of the corresponding part of the occipitotemporal cortex; hence providing a foundation for hemianopsia.

In R. H. (Figs. 9 and 10), an aneurysm of the circle of Willis just in front of the optic tract and chiasma impinged on the circulation of the right hemisphere, with swelling of the right hemisphere without actual softening or infarction, but marked herniation of the subiculum; left hemiplegia and hemianesthesia, three days later coma, and nine days later death.

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* Read at the Forty-Sixth Annual Meeting of the American Neurological Association, New York, June, 1920.

Fig. 1.—A. M., glioma with cystic degeneration in the left opercular region.

Fig. 2.—A sagittal depression of the uncus on either side marking the line of the tentorium; also moderate wedging of the cerebellum into the foramen magnum.

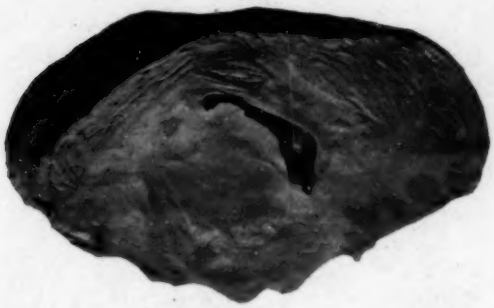

Fig. 3.—D. W., general extent of the glioma bulging into the hind-brain ventricle.

Fig. 4.—D. W., hydrocephalus visible in the distention of the tuber cinereum. Change of contour of the isthmus through the glioma; marked sagittal depressions of each uncus.

Fig. 5.—Prolapsing glioma (G. McL.); herniation of the left frontal lobe under the falx. Almost the entire left uncus is displaced on the inner side of the tentorium.

Fig. 6.—M. B., metastatic cancer and cyst taking up the mesial edge of the hemisphere. The lateral ventricles are somewhat compressed. The fornicate gyrus is pushed under the falx beyond the median line.

Fig. 7.—A ridge of herniation of the subiculum of the cornu ammonis to either side of the pineal region; compression, on the right side, of the posterior cerebral artery to the calcarine region; complete reduction of the calcarine and fusiform gyrus cortex on the right side, in contrast to the normally wide cortex on the left.

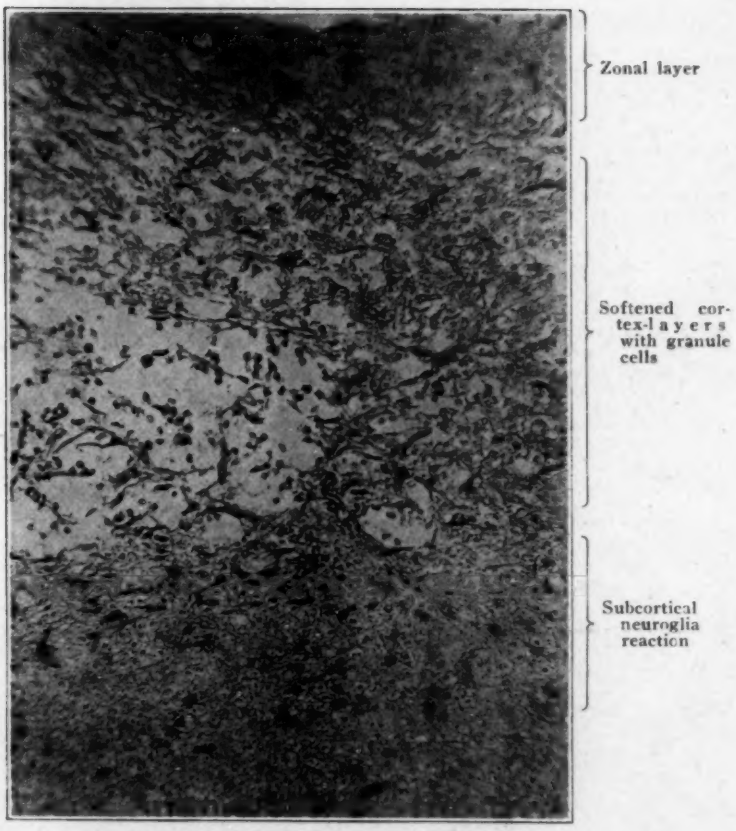

Fig. 8.—Calcarine cortex of Figure 7 with complete collapse of the cortex layers but preservation of the subcortex.

Fig. 9.—Swelling of the right hemisphere (to the left in the illustration).

Fig. 10.—Hemorrhage from aneurysm in front of the optic chiasma; interference with the circulation in the right Sylvian artery; tentorial lines of the uncus and marked herniation of the subiculum (to the left of the midbrain).

Fig. 11.—M. W., cancer metastasis in the right postcentral gyrus (to the left in the illustration).

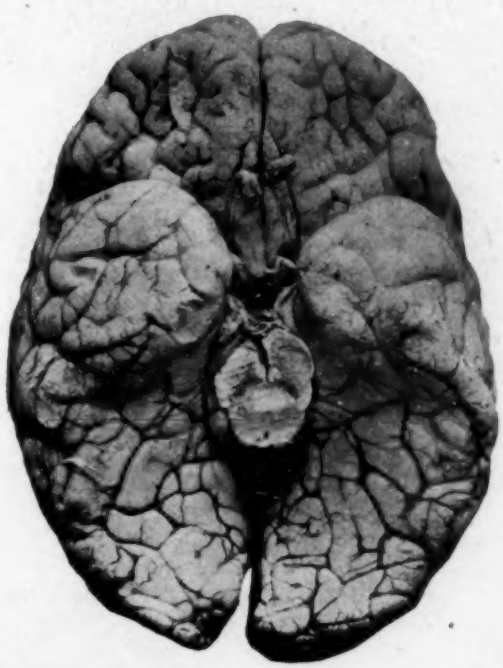

Fig. 12.—M. W., tentorial lines of the uncus and plain herniation of the subiculum along crus and midbrain.

Fig. 13.—W. H. J., character of the cystic glioma extending from the striatum and frontal region through the temporal lobe to the ventral lip of the calcarine cortex (Fig. 16).

Fig. 14.—W. H. J., subtentorial herniation of the right uncus (to the left in the illustration) similar to that seen in Figure 5.

Fig. 15.—Continuation of the uncus hernia into a thick protrusion the entire length of the subiculum without occluding the posterior cerebral artery.

Fig. 16.—W. H. J., occipital horn and calcar avis. Cystic glioma of the basal lip of the calcarine. Prominent ridge of subiculum region forming a subtentorial herniation.

Fig. 17.—W. H. J., herniation into the foramen magnum.

Fig. 18.—B. H., cancer metastasis of the left cerebellar hemisphere.

Fig. 19.—B. H., extent of the cancer.

Fig. 20.—B. H., distortion of hindbrain ventricle by the cerebellar cancer.

Fig. 21.—B. H., infratentorial distortion of the midbrain; remarkable supratentorial distention of the splenium and dislocation of the limbic lobe.

Fig. 22.—Foraminal wedging in case B. H.

Fig. 23.—Child of 10 months; convulsions. Moderate cerebellar wedging in brain swelling.

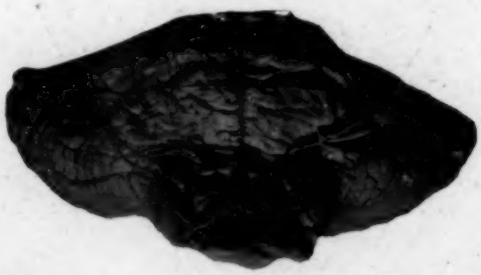

Fig. 24.—Marked wedging of the foramen, connected with lumbar puncture.

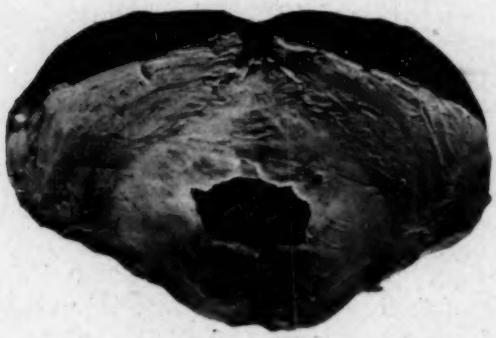

Fig. 25.—Distention of hindbrain ventricle in foraminal wedging.

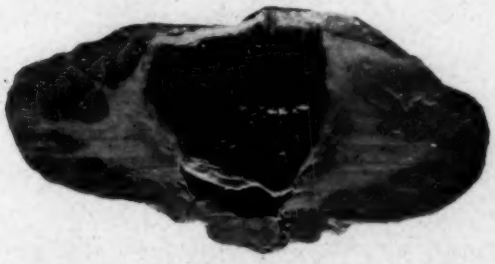

Fig. 26.—Cerebellar cyst causing foraminal herniation and partial compression of vertebral artery with bruit.

Fig. 27.—Herniation of cerebellum leading to compression of the vertebral arteries.

In M. W. (Figs. 11 and 12), an hemorrhagic cancer metastasis in the right postcentral gyrus led to subtentorial herniation and to wedging of the cerebellum.

An unusually great subtentorial herniation occurred in W. H. J. (Figs. 13 to 17), with a cystic glioma extending from the right frontal lobe to the right occipital lobe, without, however, occluding the calcarine artery. Wedging of the cerebellum was added as a secondary consequence.

In B. H. (Figs. 18 to 22), a cancer metastasis in the inferior semilunar lobule of the cerebellar hemisphere led to cerebellar herniation into the foramen magnum (Fig. 22), and to an interesting supratentorial distention of the region of the splenium, with death eight days after a subtemporal decompression.

Moderate wedging of the cerebellum (Fig. 23) in a child of 10 months, with convulsions and brain-swelling, but no focal lesion, led to a case of marked wedging of the cerebellum, distention of the hindbrain and forebrain ventricle (Figs. 24 and 25); severe frontal headache, lumbar puncture, followed by aggravation and disturbed vision and death in three weeks.

A counterpart to the case with secondary hemianopsia is produced by a case of my associate, Dr. Charles Bagley. The patient presented frontal headaches, convulsions, occasional staggering, but a vascular bruit over the entire cranium. A cerebellar cyst (Fig. 26) had produced a foraminal wedge which carried the vertebral arteries about 5 cm. below the foramen (Fig. 27).

CONCLUSION

Herniation in brain tumor and brain swelling occurs under the falx, under the tentorium and into the foramen magnum.

In the presence of pressure symptoms, we must remember that hemianopsia or vascular bruit may be a symptom of herniation.

A suspicion that herniations of the subiculum might be responsible for the sclerosis of the cornu ammonis in epilepsy through the effects of transitory subtentorial herniation is not, so far, corroborated by the present series.

CONTRIBUTION TO THE HISTOPATHOLOGY OF "PSEUDOTUMOR CEREBRI"

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INTRODUCTION

In 1904, Nonne [1] reported a series of cases which he tried to combine into a clinical entity. This was characterized by a more or less chronic development of the picture of brain tumor, with or without localizing signs, and without discoverable etiology. The patients usually recovered, either spontaneously or after treatment with mercury and iodids. If death occurred, no adequate macroscopic or microscopic lesions could be found. Nonne went so far as to claim that in some cases no microscopic pathology was present.

Nonne's publication was followed by numerous reports of similar cases in some of which the symptom-complex in question was referred to as "die Nonnesche Krankheit" [2] or "das neue Krankheitsbild." [3] Prior to Nonne, a number of similar cases were reported by Quincke [4] under the name of serous meningitis.

It was well known that various pathologic conditions could simulate brain tumor, but the idea that at necropsy no pathologic changes or an unknown pathologic lesion might be found met with widespread skepticism. At the present time, such an opinion is practically abandoned, although Nonne in his last word on the subject [5] still maintains that such cases exist.

As foundation for this opinion, there could be found in the literature only two cases: Case 11 in Nonne's [1] series, and case 9 in Reichardt's.[6] Of these, Reichardt's bears clinically no resemblance to brain tumor,

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1. Nonne: Ueber Fälle vom Symptomenkomplex von Tumor cerebri mit Ausgang in Heilung, Deutsche Zeitschr. f. Nervenheilk. **27**:169, 1904.

2. Oppenheim and Borchardt: Zur Meningitis serosa chronica circumscripta (cystica) des Gehirns, Deutsche med. Wochschr. **36**:57, 1910.

3. Finkelnburg: Ueber Pseudotumor cerebri, Zentralbl. f. d. Grenzgebiete d. Med. u. Chir. **15**:515, 1912.

4. Quincke: Ueber Meningitis serosa, Samml. klin. Vortr. **67**:655, 1893 (Innere Med., no. 23).

5. Nonne: Der Pseudotumor cerebri, Neue Deutsche Chirurgie **10**:107, 1914 (Die allgemeine Chirurgie der Gehirnkrankheiten, II Teil).

6. Reichardt: Zur Entstehung des Hirndrucks und anderer Hirnkrankheiten (u. s. w.), Deutsche Ztschr. f. Nervenheilk. **28**:306, 1909.

but was a straightforward case of catatonia. In Nonne's case, one cannot help feeling that if the microscopic examination, which was very inadequate, had been more thorough, some changes would have been found. Cases studied microscopically with care have always shown pathologic changes. Only six such examinations have been recorded and they will be briefly reviewed.

REVIEW OF CASES WITH HISTOPATHOLOGIC REPORTS

CASE 1.[7]—A man, aged 20, gave a history of gradually developing frontal headache, with defective hearing on the right, defective vision, double vision and lethargy.

Findings.—Atrophy of the optic nerves was found, the sense of smell was absent, the right corneal and conjunctival reflexes were sluggish, there were hyperesthesia of the upper branch of the right trigeminal nerve, and tenderness to pressure in the right suboccipital region. Diagnosis: tumor of the posterior fossa.

Necropsy.—No macroscopic pathologic change was discovered. Microscopically, widespread leptomeningitis (not tuberculous or syphilitic) over the base of the brain, and perineuritis as well as interstitial neuritis of the optic, olfactory and right acoustic nerves were noted. There were no changes in the brain except slight perivascular infiltration in the pons and medulla. Some polymorphonuclear leukocytes were seen around the eighth nerve.

Finkelnburg [7] intimates that this is the first time a case simulating brain tumor has been properly examined. The findings we accept, but certainly the clinical picture was stretched to make it a brain tumor.

CASES 2 AND 3.—In these cases, studied pathologically by Rosenthal,[8] it is impossible to judge just how closely a tumor picture was simulated since the history and clinical examination are very incomplete.

Findings.—Stiffness of the neck, stupor, falling to the right, cerebellar gait, and optic neuritis were present in Case 2. Regarding Case 3, it is stated only that the disorder was of cerebellar nature with optic atrophy.

Necropsy.—Nissl's acute cell disease involved the cortex; there was an increase of lipoids in the ganglion cells; the glia cells were not much changed, those along the vessels contained much fat, and the blood vessels were much dilated; macrophages (Abräumzellen) with blood pigment were found; preameboid and ameboid glia cells were seen near the ventricles; there was an increase of subependymal glia fibers; the leptomeninges were slightly infiltrated with lymphocytes, blood pigment and greenish products of cell catabolism (Abbaustoffe).

The findings are those of lowgrade meningo-encephalitis with hemorrhages and reactive phenomena in the glia.

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7. Finkelnburg and Eschbaum: Zur Kenntnis des sog. Pseudotumor cerebri mit anatomischen Befund, Deutsche Ztschr. f. Nervenheilk. **38**:35, 1910.

8. Rosenthal: Histologische Befunde beim sog. Pseudotumor cerebri, Ztschr. f. d. gesamte Neurol. u. Psychiat. **7**:163, 1911.

CASE 4.—A 14 year old boy gave a history of gradually developing left-sided weakness, psychomotor dullness, occasional headaches and vomiting.

Findings.—Left-sided spastic hemiparesis, uncertain gait, and increasing stupor were noted.

Necropsy.—There were no macroscopic changes. Microscopically, there were found marked lymphocyte and plasma cell infiltration of the adventitial spaces, with hyperemia and occasional hemorrhages; numerous large nucleated epithelial cells; occasional fat granule bodies (Körnchenzellen) and rare giant cells, especially in Langhans' sense. These changes were found around the small blood vessels of the right internal capsule, the neighboring lenticular nucleus, the dorsolateral part of the pes pedunculi, both subthalamic regions, the white substance of both cerebellar hemispheres and the nucleus dentatus.

The clinical picture in this case is again not quite convincing. Choked disk is absent and there were no epileptic fits. Yamakawa,[9] who recorded the foregoing case, came to the conclusion that the changes found belong to the category of atypical tuberculous connective tissue changes.

Nonne[5] reported two further cases with histologic examination, in the first by Spielmeyer and the second by Ranke.

CASE 5.—A woman, aged 47, after a psychic trauma, had transitory attacks of vomiting and diarrhea, with pain in the left foot. Soon afterwards she had double vision and spots before the eyes. For eight months there was gradually lessening acuity of vision.

Findings.—The patient was able to distinguish only light and dark; the skull was somewhat tender to pressure over the forehead; bilateral high grade choked disk was observed.

Course.—Right decompression was performed, with resulting hernia cerebri and death.

Necropsy.—Spielmeyer found macroscopically greatly dilated ventricles. Microscopic examination revealed moderately widespread affection of the cortex with involvement of the white substance, without dependence of the affection on the blood vessels. In the cortex the fibers were badly damaged and often entirely absent over wide areas; the nerve cells showed many chronic changes, and a great increase of the glia cells (Trabantzellen). In the deeper layers of the cortex the glia fibers were greatly increased, but particularly in the white matter. The glia cells did not show the characteristics of glioma cells. They resembled those seen in secondary glia proliferation. The process involved especially the vicinity of the central fissure and the adjoining frontal region.

Spielmeyer stated (according to Nonne) that he knew of no sickness which could cause the changes found. It seems that they may be well explained by the operation, which resulted in a hernia of the brain, hemiplegia and other permanent organic lesions.

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9. Yamakawa: Eine chronische disseminierte Encephalitis unter dem Bilde des sog. Pseudotumor cerebri, Mitt. a. d. med. Fakult. d. k. Univ. zu Tokyo **10**:201, 1913.

CASE 6.—For four weeks a 35 year old man had occasional spells of vomiting, and for some time headaches which grew worse at night. Later there were double vision and spots before the eyes.

Findings.—Definite but not high grade choked disk with hemorrhages was observed, the pupillary reactions to light were sluggish and slight, the blood Wassermann reaction was positive, the pressure of spinal fluid was high, with a cell count of 25.

Necropsy.—Marked hydrocephalus internus and ependymitis granularis were present. Microscopic examination of the brain revealed cadaver changes (27 hours postmortem); infiltration of the pia with lymphocytes and an occasional mast cell; slight perivascular infiltration with lymphocytes and an occasional mast cell in the cortex, and very marked similar infiltration in the cerebellum, brain stem and basal ganglions; products of tissue catabolism (Abbauprodukte) in all regions, especially near the ventricles, where glia cells were present with homogeneous bodies containing lipoid material; widespread ependymal proliferation in all the ventricles; occasional fresh hemorrhages in the cortex, and proliferation of the intima cells without signs of fresh proliferation in many of of the blood vessels.

Ranke concluded that there was present a chronic inflammatory process, most probably syphilitic, with secondary hydrocephalus, and that the glia, ependymal and "Abbau" changes near the ventricles were probably secondary to the pressure of the ventricular fluid. From the clinical history one would rather have suspected that cerebral syphilis and not a tumor would have been found.

It will be noted from the foregoing reports, what would be even more evident if the histories of other cases not reviewed should be recorded, that full-fledged tumor pictures are rarely present. The localizing symptoms of tumor are usually very meager; choked disk is not very pronounced and often absent entirely.

REPORT OF AUTHOR'S CASE

The patient whose report follows presented a definite left cerebellar hemisphere syndrome and bilateral choked disk reaching from 3.5 to 4 diopters.

L. McN., a school girl, aged 16, was admitted to the Peter Bent Brigham Hospital (Surg. No. 11187), Sept. 19, 1919, complaining of vertigo, attacks of headaches and vomiting, poor vision, cross-eyes, weakness of left side, and instability in walking.

History.—The family history was negative. The patient's birth was normal. She was a little late in walking and her mother says that her head has always been rather large. She had diphtheria eight years ago and a mild attack of influenza last winter, but has never otherwise been seriously ill. She had no injuries and no operations. Catamenia was normal. Her habits were good.

Present Illness.—According to the patient's father, this began after an attack of influenza, November, 1918. While playing in church the notes became dim and she had a severe frontal headache which afterwards spread over the entire head, and she took to bed. The next day the headache was gone, but since then every week or so she had severe frontal headache afterwards passing

over the entire head and lasting for a day or two. Later, these headaches localized in the back of the head, and the neck became sore at times. Her father noticed that when she had severe headache she staggered in walking and was apt to go to the left, and that she could not use her left arm and leg so well as the right. The patient, however, says that she had these headaches for several months before the attack of influenza, though never so severe. The father noticed that her left eye was turned in last March, but the patient's aunt said that the eye had been crossed for some time previously; just how long is not certain. About two weeks before admission, after an interval of freedom from headaches for three weeks, lumbar puncture was performed. This caused very severe headache which has never been entirely relieved and was made worse by assuming the erect posture, so that the patient has not been out of bed since. She was not so alert as formerly and at the time of examination was very stupid. Dr. Barrett of the Charles Sanitarium made a Wassermann test of the blood and spinal fluid, which gave negative results.

Physical Examination.—The patient was well developed and well nourished. Her brow was furrowed, she moaned from time to time as though in great pain, and also yawned occasionally, and rubbed her nose. The head seemed rather large and square, but there was no cracked-pot sound. There was suboccipital tenderness on both sides, perhaps greater on the left. No dilatation of the extracranial veins or exophthalmos was present. Neck, heart and abdomen exploration proved negative. The blood pressure was 108 systolic and 58 diastolic. The skin and temperature were normal.

Cranial Nerves.—Olfactory: Odors were recognized, and there were no subjective odors.

Optic: Fundus examination revealed on both sides that the disk margins were obscure, the disk being elevated about 1 diopter. The physiologic cup was filled, the veins were slightly engorged, and there was slight perivascular streaking, but no hemorrhages. There was bilateral choked disk, but no hemianopsia to rough test.

Motor Oculi, Patheticus and Abducens: There was downward and outward squint of the right eye with ptosis of the right eyelid. The pupils were equal and regular and reacted to light but not to accommodation. Movements of the right eyeball were restricted inward and upward. There was no nystagmus.

Trigeminal: No involvement of this nerve was manifested.

Facial: This nerve was normal.

Acoustic: No positive findings of disturbance of this nerve were elicited.

Vestibular: There was no dizziness; the Romberg sign and gait were not tried.

Glossopharyngeal: There was some difficulty in swallowing, particularly during the preceding days, and the voice had changed in character. The aunt thought it was harsher than usual.

Vagus: The pulse was regular but slow. The palate was symmetrical. There had been considerable vomiting.

Spinal Accessory: The trapezius was of normal power on the right side, somewhat weak on the left.

Hypoglossal: There was no deviation of the tongue and no tremor.

Cerebrum.—Frontal: The patient had lately become very stupid and said herself that she did not take as much interest in things as formerly. At the time of examination she could scarcely be aroused. Precentral: There were no convulsions or twitchings. Postcentral: No disturbance of touch, pain or temperature sensitivity, muscle sense or sense of position was noted. Tem-

poral: There were no hallucinations of smell or taste, no uncinate attacks, and no hemianopsia. Occipital: There were no hallucinations of light and no hemianopsia. Parietal: Astereognosis was not present.

Cerebellum.—There was suboccipital tenderness on both sides, possibly slightly greater on the left. There was some stiffness of the neck and flexion caused some pain. There was no nystagmus. The Romberg test and the gait were not tried, but there was a history of staggering to the left and inability to stand with the eyes closed, the patient falling to the left. There was marked hypotonicity on the left side, more marked in the upper extremity. Holmes' wrist sign was positive, as was Holmes' finger sign also. There was marked adiadokocinesis in the left hand. Muscle sense and sense of position seemed to be fairly normal. There was marked ataxia of the left upper and lower extremities on finger to finger, finger to nose and heel to shin tests. Caloric and rotation tests were not tried.

Reflexes.—The biceps, triceps and periosteal reflexes, knee jerks and ankle jerks were elicited, being greater on the left than on the right. The corneal, epigastric, abdominal, and plantar reflexes were present, but the left plantar was very sluggish.

Vasomotor: No vasomotor disturbance was observed.

Sphincters.—The control of these was good.

Roentgen-Ray Examination.—The convolutional impressions were increased throughout the skull. The sella was not well outlined. The frontal bone appeared irregularly thin, the parietal bones were thickened and the skull in this region was irregular in outline.

Operation.—Suboccipital exploration for presumed cerebellar tumor was performed by Dr. Harvey Cushing, Sept. 23, 1919, the anesthetic being ether.

The usual cross-bow incision was made. The suboccipital bone was much thinner on the left, though there were no patches of exceptional thinning. A wide area of bone was removed. There was an exceptionally deep bony septum projecting between the two hemispheres for a depth of about 1 cm. Some difficulty was experienced in dislodging this median partition and in doing so the dura near the lower part of the field was slightly torn and cerebrospinal fluid escaped abundantly through the small opening. There being no further risk in incising the dura, this procedure disclosed two normal appearing hemispheres. It is possible that there may have been some wedging of the cerebellum into the foramen, though this was not extreme, and on pulling up the two hemispheres the calamus could be seen. The right hemisphere was larger and possibly more firm as though there might have been a tumor. An exploratory needle was inserted into each hemisphere to a depth of 5 cm. without disclosing a lesion (possible cyst). The patient took the anesthetic well, and the wound was quite dry throughout. It was closed in layers, but just before the final closure a perforation was made through the pole of the left hemisphere and the needle was inserted into the ventricle, an abundance of fluid being reached at a depth of 3 cm., evidencing therefore an internal hydrocephalus.

Postoperative Course.—The patient made a good ether recovery. Five days after the operation she was much brighter; the headaches had been entirely relieved, but the ptosis of the right eyelid persisted.

The cast was removed October 3 and the stitches taken out. The wound healed in excellent approximation, but some fluid was under the incision line.

Headaches and vomiting were completely relieved, and the patient thought that her vision was better, but there was no improvement in ability to use the left arm and leg.

The patient was up in a wheel chair October 5. Examination of the left fundus disclosed that the disk and the vessels over the disk were obscured by new tissue, the veins were tortuous, the disk was not elevated. The same conditions were noted in the right fundus, with small hemorrhage in the upper border of disk.

The patient was very well and happy until the morning of October 10. While sitting up in a wheel chair she became dizzy and faint, was put back

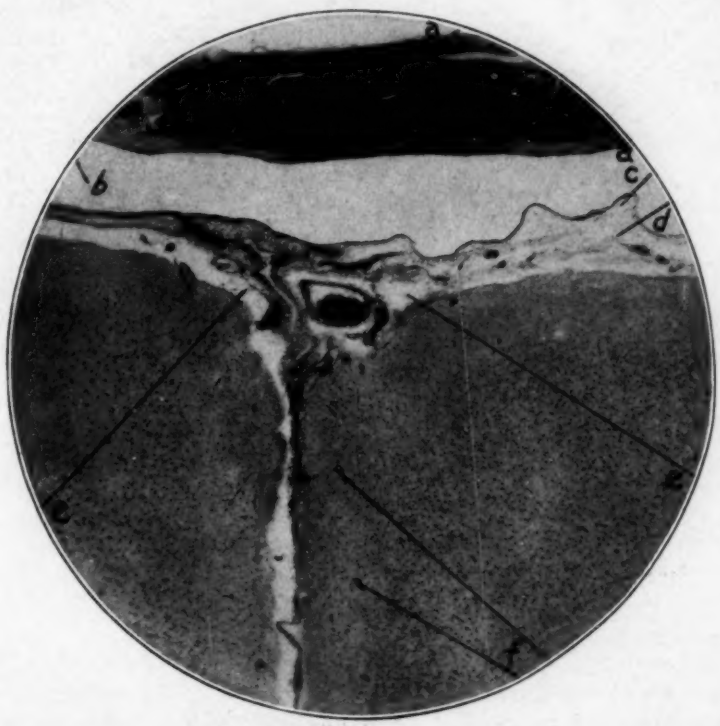

Fig. 1.—Transverse section from the motor area near the longitudinal sinus, the three membranes included. *A*, inner and outer layers of the dura, the cartilaginous mass between being very darkly stained and connective tissue strands dividing it being distinctly seen. *B*, subdural space. *C*, arachnoid membrane. *D*, subarachnoid space, greatly infiltrated, especially in the region of the sulci. *E*, so-called epicerebral space (probably an artefact, though some amorphous substance is seen within the space said to be the result of edema). *F*, small vessels. (Celloidin section, thionin stain, ×24.)

to bed and recovered. After lunch she became faint and stupid and vomited. Lumbar puncture showed that the fluid was not under tension, and perfectly clear. About 6:30, the patient had a generalized convulsion. There were clonic movements of all the limbs beginning on the left side, but she did not lose bladder or rectal control. The respiration and heart rates continued normal.

On the following day the patient had another convulsive attack about 3 a. m., followed by profuse perspiration. She was fairly bright and said her headache was better. October 16, she was very dull all day and could not be induced to talk.

October 19, there was bilateral ptosis and bilateral downward and outward squint with paralysis of convergence, both more marked on the right. The right pupil was slightly larger than the left. On the left side the reflexes were slightly exaggerated with marked hypotonicity and ataxia. The decompressed area was full, but not especially tight. No sensory disturbance was elicited. The

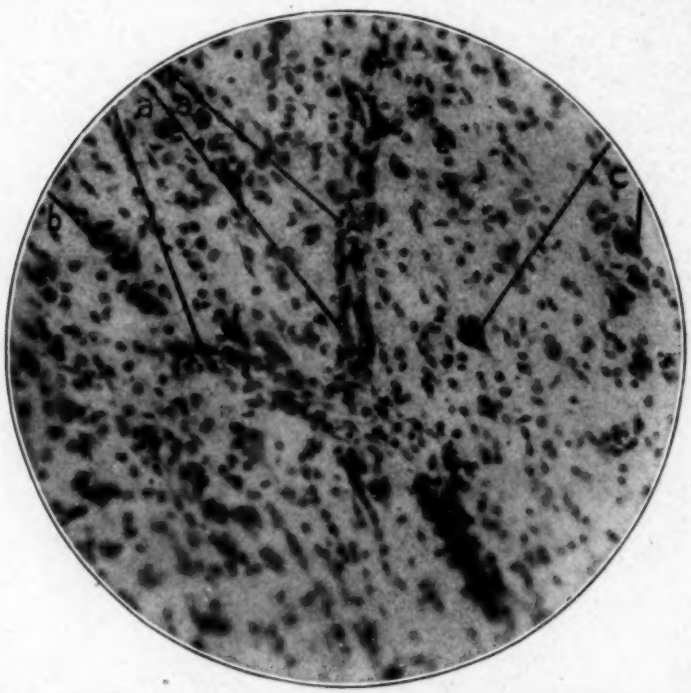

Fig. 2.—Section from the temporal lobe. The center of the picture is occupied by a branching vessel. The vessel walls appear to be infiltrated, but the numerous cells surrounding the branches are glia cells. (Use hand lens.) The rest of the picture is covered by a great number of ganglion cells and glia nuclei. Many of the former are in a state of neuronophagia (*B* and *C*). *A*, glia wall. *B*, ganglion cell surrounded by eleven glia nuclei. *C*, two other ganglion cells invaded by glia cells. (Thionin stain, ×225.) See also Figure 6.

Babinski reflex was negative. Both disks were very vascular, the veins were enlarged and tortuous and the disks elevated 3 diopters; large hemorrhages were noted in the periphery. The patient was usually very stupid. The convulsions began with twitching in the right eyelid.

This condition remained unchanged with some slight daily progression for the worse until the child's death, November 4, forty days after operation.

Presumptive Diagnosis.—Basilar tumor (unverified) with hypdrocephalus.

PATHOLOGIC FINDINGS *

Macroscopic.—The necropsy was limited to the brain, which was removed in its meninges after intracarotid injection of formalin. No macroscopic pathologic changes were present except slight hydrocephalus.

Microscopic.—Material: Blocks from the right frontal, motor, parietal, occipital, and temporal regions of the cerebral cortex, left cerebellar hemisphere, and pons, mid-brain, thalamus, and medulla were used. The material was all preserved in 10 per cent. formalin.

Methods: Celloidin, frozen and paraffin sections were stained by thionin, toluidin blue, van Giesen, Weigert's elastica stain, Alzheimer-Mann, Herxheimer's scarlet red stain, and Bielschowsky's silver method.

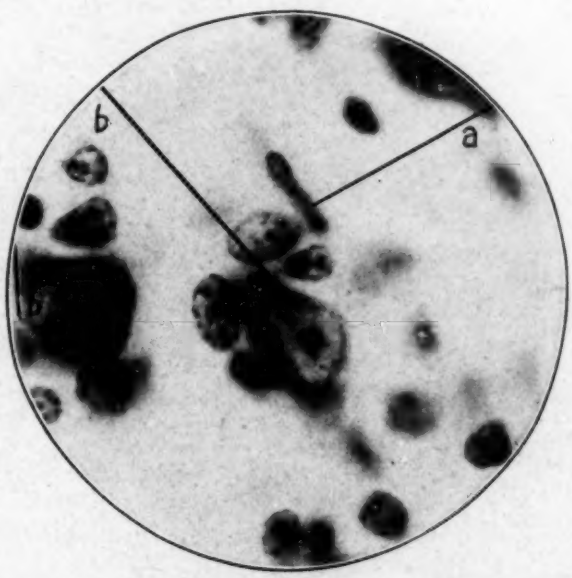

Fig. 3.—Section from the temporal lobe. *A*, rod-cell (Stäbchenzelle). *B*, two ganglion cells in a state of neuronophagia. The rest of the cellular elements are glia cells. (Thionin stain, ×1200.)

A modification of the Alzheimer-Mann (methylblue-eosin) method gave very valuable pictures on formalin-fixed celloidin material. The following procedure was used:

1. Fix in 10 per cent. liquor formaldehydi.
2. Embed in celloidin; cut 15 microns or thinner, in 70 per cent. alcohol.
3. Wash in distilled water.
4. Transfer to aqueous solution of phosphomolybdic acid, from 1 per cent. to saturated, for from one to twelve hours.
5. Pass through two changes of distilled water into

* From the Pathologic Laboratory of the Cook County and Psychopathic Hospitals, Chicago.

6. Mann's stain (eosin, 1; methyl blue, 1; distilled water, enough to make 100), for from one to twelve hours (preferably twelve).
7. Differentiate directly in 70 per cent. methyl alcohol; move constantly.
8. Transfer to 95 per cent. ethyl alcohol and mount on slide.
9. Add absolute ethyl alcohol on slide.
10. Xylol; Canada balsam (creosote may be used).

The stain comes off in the methyl alcohol in a regular fog and the methyl alcohol differentiation may be carried as far as desired. If left too long in the ethyl alcohol, the eosin will be lost, while the absolute ethyl alcohol also removes the celloidin. The latter contingency may be obviated by adding

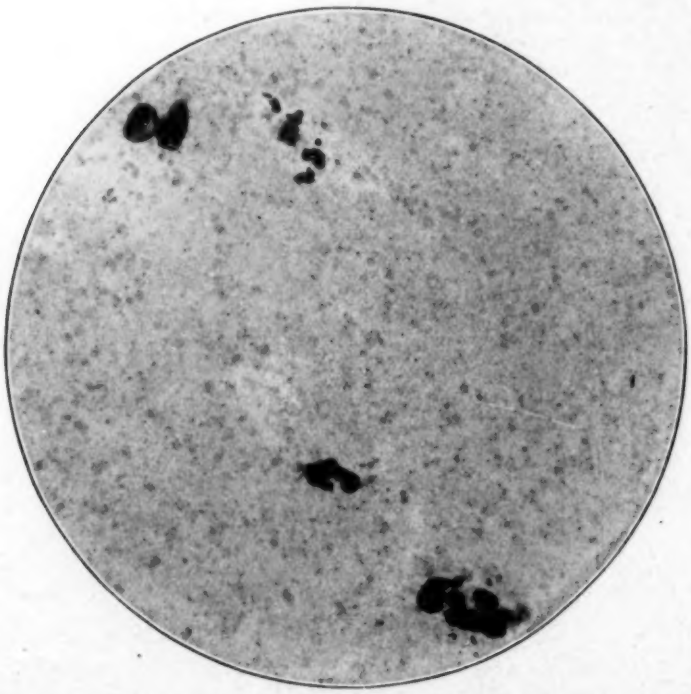

Fig. 4.—Section from the motor area. The perivascular spaces of the vessels are packed with fat (large dark globules). (Frozen section, Herxheimer's scarlet red stain, ×225.)

20 per cent. chloroform to the absolute alcohol. The mesodermal tissue, namely, the pia and vessels down to the finest capillaries, is stained a bright blue; the glia, violet to purple; the nuclear structures, dark red; the myelin, a bright pink, and the red blood corpuscles, a brilliant orange. The color contrasts are much less striking if examined by artificial light.

Meninges: Microscopically the dura showed no changes with the exception of some areas near the longitudinal sinus, consisting of almost structureless formations, containing here and there small lacunae filled each with a single cell (Figure 1). The nature of these masses was hard to determine. They were more or less uniformly stained by thionin, van Giesen, Alzheimer-Mann and Weigert's elastica stain, filled up the enlarged dural interspaces to which they were strictly confined. They resembled cartilage or newly formed bone.

The enlarged subdural space was without cellular or other contents, which, however, were abundant in the pia-arachnoid, subpial (epicerebral) and subarachnoid spaces (Figure 1). The pia-arachnoid cellular infiltration was present everywhere, but was most marked around the medulla, while over the parietal lobe the pia appeared scarcely infiltrated, but edematous.

The localization of the infiltrative elements was best studied in specimens stained by the modified Mann method, and their character was most readily determined in thionin stained specimens. These elements included lymphocytes, plasma cells, macrophages, rod cells, polyblasts, and fibroblasts. Most numerous were plasma cells and lymphocytes, mixed with erythrocytes.

Fig. 5.—Section from the motor area. The field is occupied by a great number of vessels, the majority of which are well developed. In the stained specimen are seen a great number of newly formed vessels, one of which is seen at *A*. (Weigert's elastica stain, ×225.)

In thionin stained specimens, the lymphocytes appeared as small, round, heavily stained, often almost homogeneous nuclei, with no visible cytoplasm. The erythrocytes were cup shaped, pale green bodies without nuclei. Plasma cells were quite numerous, their eccentric nuclei with spokelike chromatin and metachromatically stained cytoplasm with a clear halo alongside the nucleus being clearly discernible. In addition, a considerable number of macrophages were seen packed with blood pigment, which was also scattered freely in the distended meshes of the pia-arachnoid.

The foregoing cellular elements (lymphocytes, plasma cells, erythrocytes, and macrophages) were mixed with so-called mesothelial cells, which are large, round or oblong nuclei containing a scanty amount of chromatin and

hardly visible cytoplasm. Somewhat similar were many other nuclei with protoplasm streaming away from either end which may be classified as fibroblasts. Polyblasts with pale horseshoe-shaped nuclei, surrounded with rich cytoplasm, were everywhere in evidence, while around the vessel walls were seen sausage-shaped, oblong nuclei without visible cytoplasm, but rich in chromatin, so-called rod cells (Stäbchenzellen). Polymorphonuclear and cribriform cells (Gitterzellen) were not found.

The vessels of the pia-arachnoid appeared to be distended, but manifested no infiltrative nor other changes.

Cortex: In the cortex, parenchymatous and interstitial changes were in evidence, being most pronounced in the parietal and temporal lobes of the

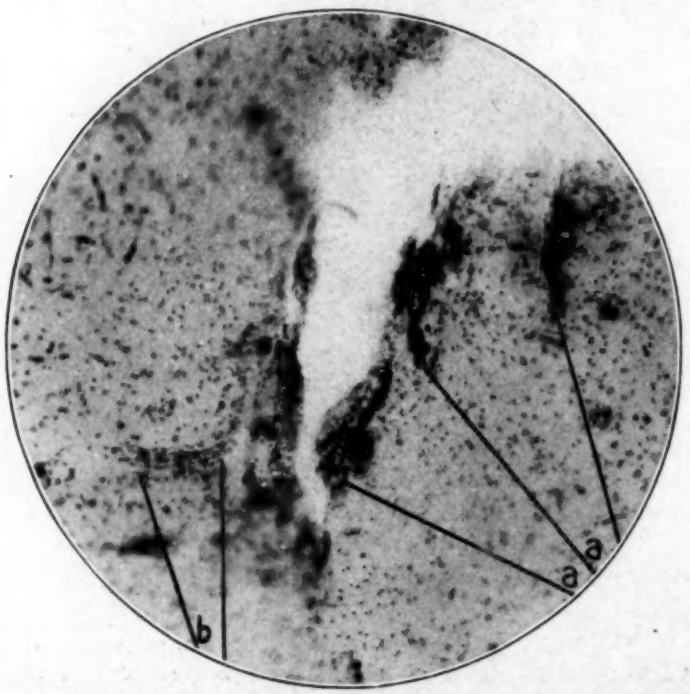

Fig. 6.—Section from the parietal lobe. Some vessels *(A)* greatly engorged and infiltrated. Other vessels *(B)* are not infiltrated, but show a beautiful row of glia cells forming a wall. (Thionin stain, ×82.)

cerebrum; the cerebellar cortex was relatively slightly involved. The parenchymatous changes predominated, and showed only in the ganglion cells, varying in degree from those of Nissl's acute cell disease to total destruction of the cells. Throughout the cortex, the majority of the ganglion cells were very poor in tigroid substance, and the nuclei were rather pale and poor in chromatin. Especially pronounced were these changes in the deeper layers of the cortex, where cell after cell could be seen surrounded by from three to ten robust glia cells (satellitosis). Though present throughout the deeper cortical regions, the satellitosis was especially marked in the parietal lobe, and attained its maximum in the temporal lobe, where it reached to the outermost layers of the

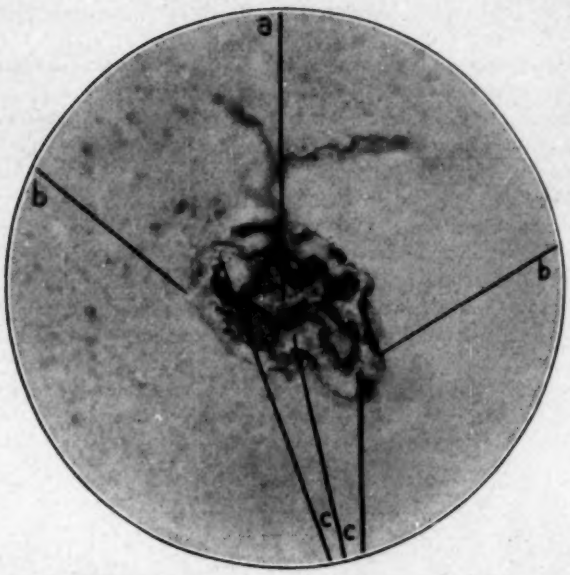

Fig. 7.—Small vein from subcortical area of temporal lobe showing the perivascular space of Virchow-Robin. It contains cellular elements which are not very distinct in the photograph. *A*, lumen. *B*, adventitia. *C*, perivascular space of Virchow-Robin. (Modified Mann's methylblue-eosin stain, celloidin section, ×230.)

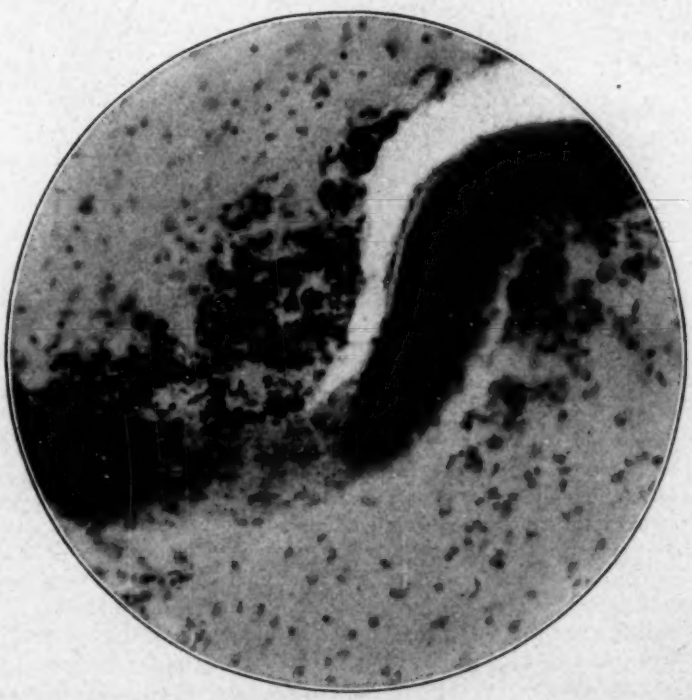

Fig. 8.—Engorged vein from subcortical region of parietal lobe surrounded by an enormous number of macrophages packed with blood pigment (black globules). (Thionin stain, ×225.)

cortex (Figure 2). In these areas, the glia cells not only clustered around but actually invaded the bodies of the ganglion cells (neuronophagia) (Figure 3). The dendrites were shrunken, heavily stained, without Nissl substance and in some places the whole cell was reduced to an amorphous granular mass. Herxheimer specimens showed that these dying cells were packed with fat, especially at the base.

The glia everywhere showed proliferative changes, chiefly in the form of increase in the number and size of cells and chromatin content of the nuclei. Many of the glia cells had an appreciable amount of cytoplasm (protoplasmic glia cells). Around some vessels, especially those where hemorrhage had occurred, a distinct glia wall (Figure 2) was formed—rows of glia cells and proliferated glia fibers. Regressive changes, such as ameboid glia cells, Füllkörperchen, etc., were absent. In the regions where neuronophagia was intense,

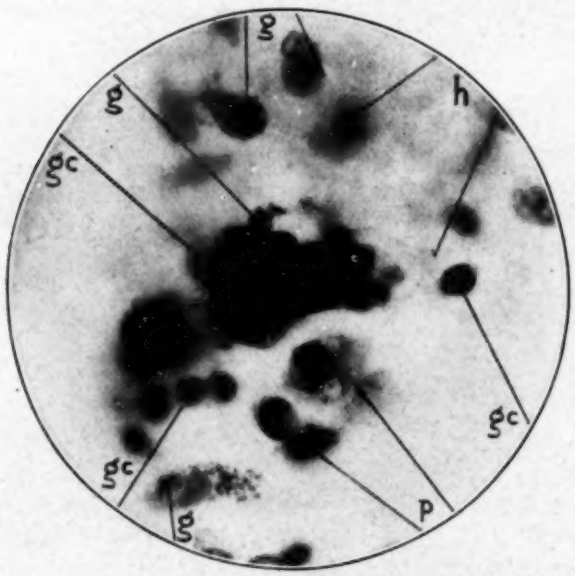

Fig. 9.—Hemorrhage, evidently of recent date. The majority of the glia cells *(G)* are packed with granules of hemosiderin. *H*, hemorrhagic focus. *G*, glia cells containing blood pigment. *GC*, glia cells, out of focus, forming a dense nest in the center and an arch of four underneath. *P*, polyblasts, the nuclei appearing dark because they are out of focus. (Thionin stain, ×1000.)

the glia cells could be seen to contain considerable fat, which was evidently transported to the blood vessels, where fat could be demonstrated in the adventitial cells. Vessels surrounded by such fat-laden cells were seen in all parts of the cortex (Figure 4), but were most numerous in the temporal lobe.

Interstitial changes in the cortex were not marked, aside from excessive vascularization. Some areas showed an abundance of vessels and capillaries, many of which were very thin and empty, with only a few endothelial cells (Figure 5). Specimens stained with Weigert's elastica stain showed a well developed elastic membrane, sometimes, but very seldom, split into several layers. Inflammatory phenomena were mild, consisting of slight dilatation of the Virchow-Robin spaces, exclusively filled with lymphocytes (Figure 6).

The interstitial changes were especially marked in the subcortical regions. Thus in the temporal lobe there was well developed perivascular infiltration, chiefly with lymphocytes, but numerous plasma cells were also present. Specimens stained with the modified Mann method showed especially well that these infiltrative cells were confined to the Virchow-Robin spaces (Figure 7). Occasionally throughout the subcortical region, but notably in the parietal lobe, a vessel could be seen surrounded by a mass of macrophages engorged with blood pigment (Figure 8), and around these marked glia fiber proliferation. The area shown in Figure 8 is evidently the site of an old hemorrhage and the macrophages are heavily laden with the products of erythrocyte destruction (hemosiderin), but at other places fresher hemorrhages may be found where minute granules can be seen in the masses of glia cells (Fig. 9).

Brain Stem: In the brain stem, the changes described in the foregoing were much less pronounced. In the thalamus was to be found slight perivascular infiltration with lymphocytes and plasma cells. Excessive vascularization was present with newly formed capillaries. Hemorrhages, evidently fresh, without reactive phenomena, were scattered about. Nowhere were there any signs of nerve fiber destruction. Occasionally protoplasmic glia cells were seen, but satellitosis and neuronophagia were practically absent. In the substantia nigra, pons, peduncles, and medulla the changes were similar, but still less pronounced, consisting chiefly of increased vascularity, hyperemia, and occasional fresh hemorrhages. Myelophages, cribriform cells (Gitterzellen) and other pathologic glia changes associated with nerve fiber destruction were totally lacking.

COMMENT

The foregoing combination of interstitial and parenchymatous changes prove that we are dealing here with a case of hemorrhagic leptomeningo-encephalitis, which clinically presented a clear-cut picture of tumor of the left cerebellar hemisphere. More than any case heretofore recorded it deserves the name of "pseudotumor."

There are some extraneous factors in this case which seem to invalidate the diagnosis. For instance, the third nerve palsy was noted as an unusual finding, but was disregarded, because the cerebellar symptomatology was so pronounced, and also because previously it had been seen in cases proved at operation to be cerebellar tumor. Again, the symptoms were stated to follow an attack of influenza, but the father also dated the squint from ivy poisoning! It may be objected that some of the pathologic findings were the result of operative trauma. This is untenable, for the subpial, parenchymatous, and other changes (neuronophagia, perivascular infiltration, etc.) were distinctly of inflammatory infectious nature. Also the pathologic changes were much less evident in the cerebellum at the site of the operation than in the temporal and parietal lobes of the cerebrum, remote from the operative field.

The combination of parenchmatous and interstitial changes will be found in any type of encephalitis, be it dementia paralytica, trypanosomiasis, lethargic encephalitis or poliomyelitis. Yet the present case

greatly differs from the foregoing types, especially from lethargic encephalitis. The presence of antemortem hemorrhages, the lack of intensive perivascular infiltrations, and what is more important, the absence of lesions in the substantia nigra, are sufficient to rule out the latter type.

The presence of antemortem hemorrhages might indicate that this was an instance of encephalitis caused by influenza, but in the absence of any reliable histopathologic reports of influenzal encephalitis it would be rash to make such a diagnosis in the present case. The majority of authors [10] (Schroeder, Redlich, and Lhermitte [11]) do not consider the cerebral lesions found in influenza as an encephalitis at all.

In view of the widespread involvement of the brain in cases of meningo-encephalitis, it is not surprising that it is difficult clinically to localize the lesion, nor that striking pathologic lesions should be found in areas not indicated by the clinical findings. In the case reported here the pathologic changes in the cerebellum were adequate to account for the cerebellar symptoms but were overshadowed by more striking changes in the cerebrum.

CONCLUSIONS

1. A case simulating a tumor of the left cerebellar hemisphere proved on microscopic examination to be a meningo-encephalitis Macroscopically the brain showed only slight hydrocephalus.

2. Cases of meningo-encephalitis may run the clinical course of a tumor of the brain even with definite localizing signs but without definite pathologic localization.

3. From a consideration of the cases properly examined, it is certain that no clinical or pathologic entity exists which deserves to be dignified by the term "pseudotumor cerebri."

4. The term "pseudotumor cerebri," if used at all, should be restricted to purposes of clinical classification without attributing to it any etiologic or pathologic significance whatever.

I am greatly indebted to Dr. George B. Hassin for constant advice and criticism, and to Dr. Harvey Cushing for permission to study this case.

10. See Bassoe, Peter, and Hassin, G. B.: Contribution to the Histopathology of Epidemic ("Lethargic") Encephalitis, Arch. Neurol. and Psych. **2**:40, 1919.

11. Lhermitte, J.: L'encéphalite léthargique, Ann. de med. **6**:306, 1919.

DYSTONIA LENTICULARIS (DYSTONIA MUSCULORUM DEFORMANS)*

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The condition generally recognized as dystonia musculorum deformans, a name given it by Oppenheim, is of special interest at this time in view of the recent significant researches concerning the functions of the basal ganglia, especially of the nucleus lenticularis. First separated as a clinical entity by Schwalbe [1] in a Berlin inaugural dissertation in 1908, given a definite nosologic classification by Oppenheim [2] in 1911, and since then the subject of a number of papers and reports, the syndrome, or disease as some would maintain, has recently (1919) received an admirable and comprehensive review at the hands of Kurt Mendel.[3] This last article renders it wholly superfluous to consider in detail the cases hitherto reported, thirty-three in number,[4] according to Mendel, which are abstracted with critical comment by him together with a minute discussion of the condition in its etiologic, symptomatologic and differential-diagnostic aspects. It is my purpose, first, to report two further cases occurring in the same family, one of which is entirely typical and the other doubtful, and secondly to consider certain aspects of this bizarre affection in its broader relations.

REPORT OF CASES

CASE 1.—*History.*—Jacob S., of Russian Jewish birth, admitted to the Massachusetts General Hospital, March 13, 1920, was originally referred to the Massachusetts General Hospital from the Peter Bent Brigham Hospital where he had been under the observation of Dr. Harvey Cushing. He was a schoolboy 11 years old, a fourth child of a family of seven. The father was

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* Read at the Forty-Sixth Annual Meeting of the American Neurological Association, New York, June, 1920.

1. Schwalbe, W.: Eine eigentümliche tonische Krampfform mit hysterischen Symptomen, Inaug. Dissert., Berlin, 1908.

2. Oppenheim, H.: Ueber eine eigenartige Krampfkrankheit des kindlichen und jugendlichen Alters (Dysbasia lordotica progressiva, Dystonia musculorum deformans), Neurol. Centralbl. **30**:1090, 1911. (Lordosis, "dromedary gait," combination of hyper- and hypotonia important; not a neurosis, four cases.)

3. Mendel, K.: Torsionsdystonie (Dystonia musculorum deformans; Torsionsspasmus), Monatsch. f. Psychiat. u. Neurol. **46**:309, 1919. (Monographic article with detailed report of previous cases and two additional ones.)

4. Mendel has evidently overlooked a number of cases reported in America, notably six by J. R. Hunt, published in 1916; see bibliography.

46 years old and the mother 44. Both were living and in general well. The patient had a brother of 19 and a sister of 15, both of whom were well and with no evidence of nervous disorder. One sister, 13 years old, had suffered for seven years from an affection presumably similar to that of the patient. She had been bed-ridden for five years. Another sister of 8 was living and well. The mother had had no miscarriages or stillbirths and denied venereal infection and alcoholism. The birth of the patient was uneventful, and he was apparently normal during infancy. He talked early and walked at 11 months. He had nocturnal enuresis until his seventh year; he had measles in 1912 and whooping cough the following year, with no complications. He began school in his seventh year and made normal progress, getting on well with the teachers but not so well with his companions. A year ago he had had tonsillitis and this year influenza. He had had no other infections or febrile diseases. He had complained of occasional headaches, usually associated with gastro-intestinal disturbances. The pain was usually frontal and temporal, beginning over the right or left eye and spreading backward over the sides of the head. It should, perhaps, be described as discomfort rather than actual pain. There had been no blurring of vision, no deafness or ear discharge. He had had no vertigo, except when nauseated. The cardiorespiratory system had shown nothing abnormal. He had had no dyspnea, precordial pain, cough, sputum, hemoptysis or fever. Several times a year his intestinal tract had been upset, associated with a watery diarrhea. He had had occasional attacks of vomiting. The skin had shown a tendency to sweating in those parts which were in muscular movement. He had had no eruptions or loss of hair. His weight had varied between 50 and 60 pounds. His habits as regards food, hours of meals and general hygiene had been, on the whole, good.

The disorder of motion began in July, 1916, with slight spasm of the right arm. He began to use his left arm instead of his right. The spasm was most marked when he attempted to use a pencil. The arm appeared weak, but he did not drop objects. The spasm gradually spread to the shoulder and then began to involve the left shoulder, passing finally to the arm. Two years later his left leg began to be affected by muscular spasm and last winter his right leg was affected in a similar way although he was still able to walk. These spasmodic movements, described later, became progressively worse so that since February, 1920, he had been unable to go out alone though he still managed to get about somewhat in the house.

Physical Examination.—This revealed a normal child except for the motor disorder which entirely dominated the clinical picture. He was sufficiently well developed and mentally was bright and responsive. He talked well and showed an intelligent interest in the situation quite beyond his years. The skin, mucous membranes, chest, heart, lungs, genital organs and abdomen, showed absolutely no abnormality. The blood pressure, systolic was 105, and diastolic, 60.

The electrical examination of the muscles gave normal faradic and galvanic response (left arm tested) both direct and indirect. The galvanic response was quick with no indication whatever of muscle or nerve degeneration. The white blood cells numbered 6,800; differential count: polymorphonuclears 74 per cent., lymphocytes 24 per cent., large mononuclears 2 per cent. The red cells and platelets were normal; hemoglobin 75 per cent.; fasting sugar 0.07 per cent.; blood calcium 12.2 mg. per 100 c.c. of plasma. The guaiac test was normal. The urine on three examinations showed varying specific gravity from 1.010 to 1.030, without sugar, albumin or other abnormal constituents.

Examination of the Nervous System.—This revealed unequal pupils, the left larger than the right; normal light and accommodation reaction; visual fields normal; slight nystagmoid movements on lateral fixation, not on direct fixation; no diplopia or paralysis of ocular muscles; fundi normal; Rinné test positive and in general hearing not affected; facial nerve normal. The fifth nerve was normal both in its sensory and motor distribution. The tongue protruded in a straight line; it was not coated. The teeth were imperfect; they were badly neglected but well placed and of good form. The palatal arch was low. The tonsils were small; palatal reflexes normal; there was no difficulty in deglutition. The arm reflexes were difficult to obtain on account of constant involuntary movements; they were not increased. The dynamometer showed the right hand grasp weaker than the left. There was no pain over the nerve trunks, no astereognosis, loss of position sense or other disturbance of sensation. The abdominal reflexes were present and equal; the cremasterics were not obtained. Knee jerks were equal and normal. There was no ankle clonus and no Babinski response. Here also the exact determination of the reflex condition was difficult to obtain on account of continuous muscular spasm. (It should be said that observations made at different times by different observers gave somewhat different results. It is, however, entirely fair to assume that the reflex conditions are essentially unchanged.) There was no muscular atrophy. The boy was of slender build but beyond the fact that some of the muscles were small they could not be regarded as in any sense abnormal. A conspicuous feature was variation in muscular tone. Some muscles were firm and elastic, showing their contours, others were flabby and toneless. In the former group were in general included the leg muscles with the exception of the tibials and peronei; both the flexors of the knee and the opposed extensors were tonic. The extensors of the hip seemed to be in greater activity than their antagonists. The triceps were tonic and opposed to the atonic biceps. The flexors of the wrist tended to overcome the extensors and at the shoulder both the deltoid and pectoralis major were tonic. The left sternomastoid was more active than the right. These observations concern the boy's usual position but as soon as he moved these balances were upset and the extraordinary spasm developed until he again placed himself in a position of rest. The foregoing description of the muscle tone can, however, not be regarded as constant even in the resting state. The facial muscles showed no spasm or contortion; in fact the muscles supplied by the cranial nerves were essentially uninvolved in the general spasm. Occasionally there was a sharp contraction of the sternomastoid. Usually when the patient was examined lying quietly in bed there was no movement of the body but the right arm was held constantly in extension and extreme inward rotation with wrist and fingers sharply flexed. This arm for certain periods might be relaxed, but there was a constant tendency to return to the contracted position. In attempting to touch his nose with the index finger of the right hand the arm was flung forward over the chest with extended forearm, then the arm was violently flexed, finally reaching the nose. This movement could only be made at a high rate of speed; a slow movement never attained its object, the arm remaining extended across the chest. Not infrequently it was impossible under any conditions to flex the arm at the elbow on account of the spasm and the attempt to reach the nose was then given up. On attempting to make a movement, as for example, sitting up in bed, the head was thrown violently to one side and backward with extreme anteroposterior flexion of the spine; one shoulder was raised above the other

and fruitless attempts were made to flex the arm at the elbow. The fingers were in the meantime in a condition of flexed spasm. As he sat up in bed with his back bent as described, the legs were flexed at the hip and the lower legs very sharply flexed against the thighs. There was no marked spasm of the feet. The head was often thrown to the right with a lateral curvature of the spine. In any attempt to move the asynergy was greatly increased. There was no hypertrophy or, as stated above, atrophy of the muscles.

Gait.—The gait of the patient was beyond adequate description. He forged forward with the pelvis sharply tilted, head and chest thrown violently back in a position of opisthotonos making extreme incoordinate, choreiform or athetoid movements to retain his balance and progress. This apparently was only possible under certain circumstances by twisting the body violently, to which movement the name torsion spasm has appropriately been given. He thrust one leg far forward and twisted himself, as it were, into a further position of equilibrium. Oftentimes in accomplishing this progression the patient turned half or even completely around and took a step backward. In fact, as has been described in other cases, progression backward may be accomplished in a much more orderly fashion than forward movement. The effort with which any progress is made is exhausting so that after an attempt to walk 20 feet, more or less, the patient is ready to drop from fatigue. The patient was able to get up from a lying position on the floor without the slightest difficulty so far as muscular strength was concerned. He showed none of the weakness or climbing efforts characteristic of the dystrophic patient but assumed the erect position with extraordinary rapidity and with various spasmodic and incoordinate movements such as characterized all of his motor acts.

Summary.—A boy of 11, of Russian Jewish parentage, was well until his seventh year; then there was gradual development of a spasmodic movement of the right arm, shoulder, left shoulder and left arm with subsequent involvement of the left leg and within a year of the right leg. Muscular movements were characterized by spasm alternating with relaxation giving rise to a gait characterized by torsion movements of extreme degree. Other than the motor disturbance there were no physical or mental abnormalities.

Case 2.—*History.*—Ida S.,[5] sister of the foregoing patient, aged 13, was admitted to the Peter Bent Brigham Hospital on Feb. 29, 1920, for gangrene of the foot. She was born in Chelsea, Mass., the third child in a family of five. The birth was normal and she developed without incident, walked at 1 year of age, talked at 2 years and was in no way abnormal up to the age of 6. She had had tonsillitis frequently until she was 7, when the tonsils were removed; she had whooping cough at 3, measles and scarlet fever at 11. In her seventh year what was regarded as "chorea" developed, manifesting itself first as a twitching of the neck and gradually spreading to the limbs and body. From this time on she also showed definite mental impairment and general retardation of body growth and had practically been confined to bed since her seventh year. For over two years she had not been able to talk intelligibly. From time to time she had had diarrhea with elevation of temperature .

Physical Examination.—This revealed a girl of 13 with the development of a child of 8 or 10. The most obvious disturbance was a constant choreiform

5. I am indebted to Dr. G. Horrax of the Peter Bent Brigham Hospital for this history.

movement extending over the entire body and also involving the extremities. This was not continuous but the patient remained in any one position only for a brief time. The facial expression was drawn and the muscles of the face were definitely involved in the choreic movements. She made only inarticulate sounds and appeared to be markedly retarded mentally. The head was very large for the size of the body, tended to be square in shape and had a moderate amount of coarse, brownish red hair. The scalp was dirty and there were pustular eruptions over the back of the head and right side of the face. The pupils were equal and reacted to light; the accommodation could not be determined. There was some opacity of each cornea which, however, apparently did not markedly obscure the field of vision. The patient's eyes shifted constantly from side to side to such an extent that it was impossible to determine whether or not a strabismus existed. The ears showed no abnormality. The nose was normal. The patient could open the mouth only by making extraordinary facial contortions, and she was unable to protrude the tongue. The thyroid was not enlarged. The thorax was infantile in type but expansion was good and equal on both sides. The heart showed nothing of significance beyond a systolic murmur at the base. The pulses were equal, regular, synchronous and of fair volume and tension. The systolic blood pressure was 90; the diastolic, 60. The lungs showed no involvement. The abdomen was protuberant. There was marked dilatation of the superficial veins over the abdominal wall. The liver, spleen and kidneys were not palpable. There were no masses or muscular spasm. Both arms and legs were held in a flexed position preventing full extension even by force. Both feet showed talipes equinus. The toes and fingers were retracted in flexion. Over the right toes and extending on to the dorsum of the foot there was a definite gangrene, presumably due to careless treatment before admission to the hospital. The emaciation of the patient was extreme. Her appetite, however, was good but she was extremely constipated. She had no nausea or vomiting and there was nothing definitely wrong with her gastro-intestinal tract. She was incontinent, presumably because of her mental state and her incapacity to communicate her needs. She slept only by the aid of sedatives which she had been taking for about two years. It was noted that the movements almost ceased during sleep but not wholly. The urine on admission gave a high specific gravity, 1.032; acid reaction, normal color, no albumin or sugar. The hemoglobin was 60 per cent. and the white blood count 6,040.

She was discharged March 31, 1920, after a residence in the hospital of about one month. During that time she was noisy, difficult to manage, disturbed the other patients in the ward and in general showed definite mental enfeeblement. The gangrene of the foot yielded to treatment and she was returned somewhat improved to her home.[6]

Summary.—The essential features in this case are apparently normal development up to the age of 7, then the appearance of widespread choreiform movements which uninterruptedly persisted up to the time of her death, a period of about six years. Mental failure, disturbance in speech, more or less facial contortion, extreme weakness and emaciation accompanied the choreic movements. In general, the physical examination, with the exception of intercurrent disorders, was essentially negative.

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6. This patient has since died, unfortunately under conditions which precluded a postmortem examination.

The cases are primarily of interest since they occurred in the same family, and secondarily because they are totally dissimilar in clinical manifestations. Both patients were attacked by the disorder at about the seventh year, from which one has since died. The disorder of motion in Case 1 has never involved the head, although, contrary to the description of many cases, it began in the upper extremities, whereas in Case 2, the neck was affected at the outset, and until her death, the illness was characterized by involvement of the head. The boy has retained his mental capacity to the fullest extent, and in fact shows no signs or symptoms referable to the head or cranial nerves except a slight pupil inequality, whereas the girl forthwith became demented. It is altogether probable that the two cases are familial in the sense that they imply some common defect of development but the second case cannot properly be classified as dystonia musculorum deformans as that term has come to be used. The first is a classic example of the condition described and named by Oppenheim, whereas the second represents, doubtless, a lesion in the same general area of the brain but with entirely different manifestations, a matter of importance in our final classification of the disorder. That two or more cases of a relatively definite and similar type may occur in one family is shown by the cases recently described by Abrahamson,[7] in which he had the opportunity of observing three members of one family, all presenting a disorder of motion of characteristic dystonia type. It remains clear, however, that with few exceptions the cases hitherto studied have occurred sporadically in much greater degree for example, than in Friedreich's ataxia, periodic family paralysis, or certain other definite familial diseases.

TERMINOLOGY

It is inevitable, but none the less unfortunate, that in the effort to establish the condition as a clinical unity, a variety of terms have been used, which further study has shown to be inadequate, if not wholly misleading. Schwalbe, in the first publication on the subject, described the condition as "an unusual variety of spasm with hysterical symptoms." This has the merit of vagueness, but it is altogether doubtful whether hysterical symptoms occur in the clinical picture. Ziehen[8] speaks of it as a "tonic torsion neurosis," which takes no account of the characteristic atonia, and also assumes by the use of the word neurosis that it has no structural basis. Oppenheim's terms

7. Abrahamson, I: A Case of Dystonia Musculorum Deformans (Oppenheim), J. Nerv. & Ment. Dis. **40**:38, 1913; ibid. **57**:451, 1920.

8. Ziehen: Fall von tonischer Torsions-neurose, Neurol. Centralbl. **30**:109, 1911.

"dysbasia lordotica progressiva" and "dystonia musculorum deformans" are less open to objection. Of these the latter terminology is to be preferred, but the use of the word "deformans" is misleading and inaccurate. Flatau and Sterling[9] add nothing in their "progressive torsion spasm in children," since the important element of dystonia is ignored and presumably also the affection is not confined to children. Fraenkel[10] considered the disorder allied to tics and spasmophilia and used the phrase "tortipelvis" which emphasizes only one and that an inconstant element. "Athetosis universalis," as used by Förster, is entirely inadequate since the movements are not universal and not truly athetoid in character. Very much better terms, because more general, are those used by Strümpell, "amyostatic symptom complex," or "myastasia;" by Mendel, "torsion dystonia" and by Thomalla "dystonia lenticularis." Of these Thomalla's[11] term is, on the whole, to be preferred since it describes adequately the muscular condition and suggests its anatomic location. Until our knowledge is much more exact than at present, this is as far as it is safe to go.

ESSENTIAL CHARACTER OF THE CONDITION

As one would expect from the diversity of terminology, the effort has been made to establish the disorder as a definite clinical entity. Oppenheim was inclined to this view and recently Mendel has asserted with conviction that we are entirely justified in including the cases in a special disease group and in postulating for it a specific etiology, a "morbus sui generis." Hunt,[12] Jelliffe, Thomalla and Strümpell are opposed to any such narrowing of our point of view which tends to divert investigation from the larger questions at issue. Experience has amply shown that a syndrome, without a defined pathologic anatomy and still less an unascertained etiology, should never be regarded as a "disease entity." The history of medicine is replete with errors into which a failure to recognize this fact has led. In this particular instance a review of the cases described as "dystonia

9. Flatau, E., and Sterling, W.: Progressiver Torsionsspasmus bei Kindern, Ztschr. f. d. ges. Neurol. u. Psychiat. **7**:586, 1911. (Jewish children; location: peduncles, cerebellum.)

10. Fraenkel, J.: Dysbasia lordotica progressiva; Dystonia musculorum deformans; Tortipelvis, J. Nerv. & Ment. Dis. **39**:361, 1912. (Analogy with torticollis; develops on a "tic" soil. Deficit in calcium metabolism.)

11. Thomalla, C.: Ein Fall von Torsionsspasmus mit Sektionsbefung und seine Beziehungen zur Athétose double Wilsonschen Krankheit und Pseudosklerose, Ztschr. f. d. ges. Neurol. u. Psychiat. **41**:311, 1918.

12. Hunt, J. R.: The Progressive Torsion Spasm of Childhood (Dystonia musculorum deformans), J. A. M. A. **67**:1430, 1916. (Six cases reported; valuable paper, overlooked by Mendel.)

musculorum deformans" forthwith shows the divergence in the symptomatology. A great variety of irregular muscular movements have already been described under the general category; torsion, tortipelvis, dromedary gait, athetosis, choreiform movements, tremor, propulsion, atonia, hypertonia, but none, with the possible exception of a general dystonia, can be regarded as sufficiently characteristic to meet the requirements. It is increasingly evident that to postulate this syndrome as a disease is to lose sight of the main issue, which is to determine under what conditions of altered structure or function such abnormal movements arise. It is therefore desirable to give up for the present the attempt to circumscribe the disorder further than to regard it as one of the outstanding symptom groups of lenticular or basal ganglion disease. The great numbers of transitional forms already described sufficiently demonstrate that it has small claim to clinical independence. A provisional classification is all that should be undertaken for the present, an attempt which Cecile and Oskar Vogt [13] in their paper on the Pathologic Changes of the Striatum and Pallidum have recently attempted. Little light has as yet been thrown on the condition through post-mortem examination. The changes in progressive lenticular degeneration have been adequately described by Wilson [14] and many subsequent observers. Von Economo [15] has recently presented a detailed account of the gross and histologic alterations but no unquestioned case of dystonia musculorum has yet been studied, unless we except one reported by Thomalla.[16] Even this case should be regarded as atypical for the following reasons, as Thomalla himself points out: the Babinski response was not always negative; there were associated athetoid movements. Necropsy showed marked changes in the putamen, which was reduced in size on both sides and softened. Microscopically the parenchyma of the putamen was fully destroyed. There was definite glia reaction in the meshes of which fat granule cells appeared. The blood vessels showed minor changes. The caudatus, globus pallidus and internal capsule were not involved. The thyroid was somewhat enlarged; there were

13. Vogt, C., and O.: Sitzungsberichte der Heidelberger Akademie der Weissenschaften, No. 14, 1919.

14. Wilson, S. A. Kinnier: Lenticular Degeneration, A Familial Nervous Disease Associated with Cirrhosis of the Liver, Brain **34**:295, Part 4 (March) 1912.

15. Von Economo: Wilson's Krankheit und das Syndrome du Corps Strié, Ztschr. f. d. ges. Neurol. u. Psychiat. **43**:173, 1918.

16. One of Ziehen's original patients is said to have come to necropsy with negative findings (reference not found) and more recently one of Flatau and Sterling's cases, which showed the Wilson type of degeneration in liver and putamen, although the patient clinically appeared to be a victim of dystonia musculorum (Jelliffe).

doubtful changes in the testicles and most important, alterations of the liver, similar to those described in Wilson's disease, were well defined. Regarding the liver changes, Thomalla believes that they represent a fundamental defect of development, in the basis of which a progressive degenerative process has occurred associated with secondary connective tissue proliferation.[17] Finally Thomalla concludes that bilateral athetosis, pseudosclerosis (Westphal-Strümpell), Wilson's disease and torsion spasm (dystonia musculorum) all characteristically show disturbance in muscle tone, and judging from the one necropsy of a case which clinically bore some resemblance to the latter condition, may be supposed to have a similar pathologic anatomy. For all of these conditions the appropriate term of "dystonia lenticularis" is proposed.

Strümpell takes a similar position in the use of the term "amyostatic symptom-complex" or "myastasia." Under this general term he includes extrapyramidal lesions, namely: Wilson's disease, pseudosclerosis, double athetosis, paralysis agitans, juvenile paralysis agitans and presumably dystonia musculorum. He believes it even possible to group in the same general category his pseudosclerosis and Wilson's progressive lenticular degeneration as exogenous disorders characterized clinically by tremor of wide range, disturbance of speech, dementia, pigmentation of the cornea (not observed by Wilson), often muscular rigidity with alterations of the liver (not primarily observed by Strümpell) and marked degeneration of the lenticular nucleus. The analogy with the condition clinically termed paralysis agitans naturally assumes importance.

In a recent discussion of Abrahamson's communication, to which reference has already been made, Jelliffe outlines the only tenable position which it seems possible to hold in the present state of our knowledge when he states that dystonia musculorum is simply one of the striking types of the varying clinical trend of the group of striatum syndromes, which includes also Vogt's double athetosis, Westphal-Strümpell's pseudosclerosis, Wilson's lenticular degeneration, Huntington's chorea, tuberous sclerotic idiocy and paralysis agitans. Jelliffe also insists that a matter of prime importance, as yet in its speculative state, is the association between liver and striatum changes

17. Little attention has been given to the possible rôle of the cerebellum in relation to the condition of dystonia. Flatau speaks of the possibility of the peduncles and the cerebellum being involved, but no systematic attempt has been made, either theoretically or postmortem to determine this point, in spite of the recognized part played by the cerebellum in the maintenance of muscle tone and coordination.

with the possibility, which the future holds out, of arriving at some sort of an understanding of the vegetative control of the muscle metabolism through the possible striatum connections.

CONCLUSIONS

The final question of diagnosis of dystonia musculorum loses much of its force if we are to consider it as a symptom group merely and not as an entity, as Mendel among others insists. As the number of reported cases grows it becomes increasingly evident that transitional forms are far outnumbering those which conform to an assumed definite type with the natural result that nothing is gained by an attempt to establish its claim as a separate "disease entity." Certain conclusions are, however, justified.

1. A type of motor disorder exists sufficiently definite to demand a provisional classification, characterized by combined hypertonia and hypotonia (dystonia) giving rise to a distinctive form of gait and motor disturbance in general.

2. Although occurring with frequency in children of the Jewish race, it is not confined to that race, nor does it always begin in childhood.

3. It is sporadic, rather than truly familial, though in a few instances more than one case has appeared in the same family (Schwalbe, Bernstein, Abrahamson, Taylor, one case atypical).

4. It is not to be sharply differentiated from various other forms of motor disorder of presumable extrapyramidal origin.

5. It is due to structural alterations of the striatum (lenticular nucleus).

6. It has no defined pathologic anatomy, but its association with alterations of the liver is probable.

7. The etiology remains wholly obscure beyond the probability of congenital defect.

8. For the present, the most suitable term both in its clinical and pathologic implications is dystonia lenticularis.[18]

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THE CHRONICITY IN LETHARGIC ENCEPHALITIS *

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During the past eighteen months over two hundred cases of lethargic encephalitis have come under our care, chiefly in Mount Sinai Hospital and in consultation at the Jewish Hospital, Brooklyn. The febrile stage of most of these patients lasted from ten to twenty days. When that stage was passed and convalescence was well established, they were discharged from the hospital. As a visiting physician watching the passage of these patients through our intern service, I at first thought lethargic encephalitis was an infectious disease of sudden onset and of rapid course which, if terminating happily, would be followed by a less rapid but complete recovery. The cases of some patients undoubtedly conformed to this view; others, in which the acute stage differed in no way, halted in their recovery and were forced to return to the Outpatient Department for further treatment. Still other patients survived the acute phase only to enter a chronic progressive stage. With every month that passed, more of these unfortunates returned. The early optimism I enjoyed quickly perished, and I learned to dread this disease, so often fatal, not infrequently inflicting permanent damage on those who survived it, and sometimes bringing in its train progressive functional deterioration.

No one is qualified from personal experience to give a definitive characterization of lethargic encephalitis, and least am I, for my experience with this disease has been gained chiefly among the racial group of the Jewish community of New York City and its neighborhood. Each of us has seen his little section of the epidemic and from it has drawn his own conclusions, which are neither the first nor the last word, but merely a small part of the general truth. It is, therefore, undesirable at this stage of our ignorance to risk a permanent distortion of our view of the disease by unduly stressing either local or personal experience. Hence, without pretence to completeness and without attempting statistical presentation, I shall concern myself merely with a brief description of some of the chronic types and persisting consequences of lethargic encephalitis.

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* Read at the Forty-Sixth Annual Meeting of the American Neurological Association, June, 1920.

THREE ELEMENTS IN LETHARGIC ENCEPHALITIS

We distinguish three elements in the disease: (1) the disease agents; (2) the effect of these agents on invaded tissues, and (3) the physiologic and psychologic consequences of these morbid effects. We know that the agents sometimes actively persist for periods far exceeding the usual twenty day limit. We know further that these disease agents sometimes produce irreparable destruction in nerve and vascular tissues and perhaps in the glandular tissues. Such permanent structural changes, in so far as they are definitive and invariable, can scarcely be classified as chronic; for the word chronic is used habitually to connote not merely an enduring, but also an active, change or process. Nevertheless, permanent mechanical defects in the structure of the body, although themselves invariable, may be followed by progressive functional deterioration to which the term chronic can be applied. And, moreover, the deterioration of a function, even if not itself progressive, may entrain a gradual disorganization of an associated function. Hence, the chronicity of lethargic encephalitis may have its origin in any one of the three elements I have distinguished: in the disease agents, in the structural alterations these produce, and in the physiologic consequences of these alterations.

Disease Agent.—The persisting disease agent may give rise to a continuous or remitting fever. The point at which a protracted infection merges into a chronic infection, can be determined only by the application of arbitrary time standards, about which at present there is no conventional agreement. The fever is probably due to the tissue response to the organism itself. When the fever finally subsides organisms may still survive in sheltered sites, where, acting with greatly reduced virulence, they may insidiously produce progressive lesions; or, if the organism itself becomes emasculated, so that it lacks the power to destroy or to injure tissues, a toxicity may still persist of a potency sufficient to cause gradual cellular damage or death and to excite in and around the affected cells the repair reactions consequent to such accidents. For instance, Buzzard has shown that in late cases of lethargic encephalitis atheromatous changes occur in the blood vessels of the central nervous system. The deposit of lime salts does not occur in healthy tissues. In the cells in which lime salts are found, degeneration has preceded its deposit. The calcification is, so to speak, compensatory to failure of the natural repair processes of the degenerated cells. We do not definitely know, however, whether the toxin which produces the vascular degeneration is derived directly or indirectly from the organism of lethargic encephalitis. As the endocrine glands are implicated in this disease, many abnormal chemical changes accompany it. These chemical aberrations affect all the cells of the

body, but the calcareous degeneration is found chiefly, if not solely, in the blood vessels of the central nervous system. It is, therefore, unlikely that autogenous chemical changes are alone responsible for the slowly developing vascular defects. In other words, it is at least probable that there exists a chronic and toxic type of the disease.

Structural Alterations.—The structural changes which endure occur in the highly organized cells—nerve, vascular and glandular cells. In the present state of our knowledge, we deduce the nature of the anatomic changes in these cells only from the alteration in the physiologic conduct. Some of these physiologic alterations are not permanent. Their disappearance presumably depends either on the absorption of inflammatory exudates or on the gradual extinction of the disease agents, permitting the functional recovery of cells which were not vitally damaged. As our observation of these physiologic changes has lasted at most only eighteen months, it is possible that changes which we might now deem permanent may yet disappear; that changes which now seem stationary may later become progressive; and that changes which are now progressing may hereafter become stationary or even retrogress. In other words, we are not yet in a position to undertake the defining and classifying of chronic types of lethargic encephalitis. All that our present knowledge justifies is the recording of the signs that tend to persist. I have collected these persistent signs, and for convenience present them in the groups, somatic, endocrine and mental.

Physiologic and Psychologic Consequences of Alterations.—The persistent somatic signs are fatigability, diplopia, blurred vision, optic neuritis and optic atrophy, faulty accommodation, nystagmoid movements in extreme lateral positions and slight differences in the width of the palpebral apertures, lack of facial expression, tongue tremors and deviations, peripheral palsies, a pseudo-Parkinson syndrome, fine and coarse tremors of the extremities, a hemiplegic spastic attitude of the upper extremity, dragging of the leg and increased resistance to passive movements with exaggerated deep reflexes, propulsion and retropulsion, unsteadiness and titubation, careful, short or unequal steps and other disturbances of gait and attitude, chorea, athetosis, dystonia, epilepsy, aphasia, all varieties of sensory disturbances and a transverse myelitis syndrome which may lead to death from decubitus or pyelonephritis.

Among the endocrine signs are obesity, menstrual irregularity, especially amenorrhea, Sergent lines, dermography, von Graefe, Moebius and similar eye signs, hyperhidrosis and anhidrosis, polyuria, low blood pressure, persistent slow pulse and tachycardia, either constant or paroxysmal, labored breathing and asthma, impotence and medullary crises.

Among the mental symptoms, those observed during the acute stage of the disease may long persist in a mitigated form. If the disease has been acute, especially if meningo-encephalitis signs occurred, the patient may remain as if stunned for months. Frequently an amnesia exists during part of or the whole period of the disease. It may even be retro-active. Some patients confabulate to fill in the memory gaps, especially such as developed a confusional psychosis of the Korsakoff type. Patients are seldom desirous of leaving the hospital. When they return to their homes even those who were quiet while in the wards may become peevish and complaining, so that often the family begs us to take the patient back. This irritability may be combined with wilfulness and anxiety. Part of this mental state arises from the belief of the patient and of his friends that death has been evaded only by a miracle. Sometimes depressions and hypomanias develop; at other times hebephrenic episodes are seen; and even fully elaborated psychoses may appear, which the family or personal history leads one to suspect are due to hereditary tendencies which the infection has brought out. Many weeks or even months usually elapse before these patients return to work, for they are timid, fearful and tired, and unable to apply themselves. To their incapacity is added a marked emotionalism. They linger in bed far into the day, and when they arise they sit about unoccupied and vaguely restless. Some are definitely hysterical; many acutely neurasthenic. The uncontrollable laughing and crying of the convalescent case of lethargic encephalitis recalls the emotional instability seen in multiple sclerosis and in pseudobulbar palsy.

Children show the same mental defects as adults. Some, however, in whom the disease began with insomnia, for months remain drowsy by day but wakeful and irritable at night.

Besides these mild forms, severe mental disturbances may persist or develop. I have seen both grave delusional and confusional psychoses, as well as a form of amentia resembling dementia paralytica (except that grandiose ideas are absent).

In this brief enumeration of such symptoms and signs of lethargic encephalitis as are remarkable for their persistence, I have not attempted to separate the stationary from the progressive, or the persistent from the permanent. In some cases, although the lesion may be permanent, the physiologic consequence may doubtless be adequately compensated and the functional disturbance may disappear. In other cases compensation may be imperfect, or it may be lacking and a constant and invariable functional disturbance persist. And if, as in the case of the internal secretions, the affected function belongs to a confederacy of functions, the whole confederacy may be deranged and thus indirectly a progressively increasing defect develop. This effect

of implication of associated functions is best seen, not in the physiologic but in the psychologic, disturbance. Heredity, environment and age may enhance or diminish the mental defect and may determine its development, its persistence or its disappearance. The factors in the chronicity of the disturbance of lethargic encephalitis are thus not merely the disease agent and the resistance of the person to this organismal invasion, but also the general functional integrity of the individual and the nature of his environment.

CHRONIC AND PROGRESSIVE FORMS OF LETHARGIC ENCEPHALITIS

Cases of protracted and imperfect recovery, as well as cases in which the deterioration of particular functions progresses, are in many instances to be considered not as chronic types of lethargic encephalitis, but as examples of the effect of the disease on persons of certain types or circumstance. In some instances such cases undoubtedly merit classification as chronic types. Thus, we may distinguish as a separate type the group of chronic cases which resemble Parkinson's disease. Another type comprises the chronic choreas; still another, the progressive dystonias and a fourth, the cases of progressive myelitis which in my experience usually end fatally. Other chronic and progressive forms may have been observed which are not within my experience. I shall not attempt to describe these four types, and I shall only mention without discussing the noteworthy fact that multiple sclerosis is not a sequel to lethargic encephalitis.

Editorial

RECENT ADVANCES IN NEUROLOGY OF THE PERIPHERAL NERVES

The late war provided an inexhaustible fund of experience in neuropathology. The study of the material afforded by the cases of injuries to the peripheral nerves has been especially fruitful. The research in the neurologic departments of the various armies has demonstrated that anatomic and physiologic ideas current prior to the war were inadequate to explain many of the phenomena associated with injuries to the peripheral nerves.

Compared with the neurologists of the other armies, those of the American army were afforded but a meager opportunity for the study of neuropathology. The few months in which we were actively engaged in warfare, with the necessity for rapid and efficient medical function, did not permit the fullest organization of neurologic units for consistent research of injuries to the nervous system in the cases observed in France. On their return to the United States, the patients with peripheral nerve injuries were assembled in a number of special hospitals where they were closely studied and, when necessary, treated surgically.

The attempts to establish syndromes to differentiate cases of anatomic interruption, compression, irritation, dissociation and recovery, chiefly by M. and Mme. Déjerine and M. Mouzon,[1] have not resulted in any definite conclusions. The American school agrees with many of the French workers that, clinically, anatomic and physiologic interruptions are indistinguishable. Valuable indications of severe lesions have, however, been established (Meige,[2] Pittres,[3] Athanassio-Benisty.[4]).

Research in the fields of motion and sensation has explained certain heretofore confusing observations. Supplementary motility was recognized by Swan in 1834. Much of the knowledge concerning this

1. Déjerine, J. J.: Déjerine, Mme., and Mouzon, J. Presse méd., May 10, July 8 and Aug. 30, 1915.
2. Meige: Rev. Neurol. **23**, April-May, 1916.
3. Pitres: Rev. Neurol. **23**:477 (April) 1916.
4. Athanassio-Benisty: Clinical Forms of Nerve Lesions and the Treatment and Repair of Nerve Lesions, Military Medical Manual, London, 1918.

type of movement, which is often responsible for a failure of recognition of the severity of a lesion, was furnished by Duchenne, Beevor and Sherren. Since the war, the importance and common occurrence of these movements has been emphasized by Claude,[5] Athanassio-Benisty, and in this country by Wood,[6] Ingham and Arnett[7] and Pollock.[8]

In the field of sensation, it is notable in the work of the Allies that, whatever their observations may have been, there is a tendency to concur with the theories of Head. Contributions to sensory studies have been of two types. First, such as detail the sensory distribution of peripheral nerves, notable among which is the work of Stopford,[9] and in this country, Hamilton[10] and Grimberg;[11] second, investigation of the possible existence of separate fibers for epicritic and protopathic sensibilities. These have been studied chiefly in the United States. The problem has been attacked from two angles; first, by Cobb,[12] who has shown, by employing painful stimuli with low degrees of pressure of known quantity, that the areas of tactile anesthesia and analgesia are coextensive; second, by Pollock,[13] whose work tends to show that the early and dissociated return of pain sensibility to higher degrees of pressure is not due to an unphysiologically rapid regeneration of protopathic fibers, but to assumption of function of overlapping nerves. A sign of some confirmatory diagnostic value (Tinel[14] has been added.

In common with other observers, the American neurologists have found electrical reactions to be disappointing as absolute indications of the severity of the lesion. Attention has been directed by the English school to the practical employment of measured shocks from a condenser to determine the degree of injury and regeneration. The importance of coexistence of vascular and joint lesions has been emphasized. The French school has called attention to the occurrence of physiopathic disorders which require differentiation from injuries of the peripheral nerves. Only a negligible number of these so-called

5. Claude: Rev. Neurol., April-May, 1916.
6. Wood: Arch. Neurol. & Psychiat. **2**:532 (Nov.) 1919.
7. Ingham and Arnett: Arch. Neurol. & Psychiat. **3**:107 (Feb.) 1920.
8. Pollock: Arch. Neurol. & Psychiat. **2**:518 (Nov.) 1919.
9. Stopford: Anatomy, October, 1918.
10. Hamilton: Arch. Neurol. & Psychiat. **3**:277 (March) 1920.
11. Grimberg: Arch. Neurol. & Psychiat. **2**:338 (Sept.) 1919.
12. Cobb: Arch. Neurol. & Psychiat. **2**:505 (Nov.) 1919.
13. Pollock: Arch. Neurol. & Psychiat. **2**:667 (Dec.) 1919.
14. Tinel: Presse méd. **23**:835 (Oct. 7) 1915.

"congealed hands" have been observed by the American neurologists, who agree with Roussy and Lhermitte [15] that they are functional in character as opposed to the view of Babinski.[16]

One of the most important investigations has been directed to the study of intraneural localization or peripheral nerve topography. The specificity of localization of nerve fasciculi was first emphasized by Stoffel. During the war, Marie, Meige and Gosset [17] were able to confirm this work, with certain modifications, by intraneural electrization. M. and Mme. Déjerine,[18] studying partial lesions the locations of which were confirmed by operation, obtained charts of intraneural localizations of a character somewhat varied from the preceding investigators. In this country a notable contribution to this subject appearing in the September issue of the ARCHIVES OF NEUROLOGY has been made by Kraus and Ingham.[19] Their investigations were a continuation of the work of Marie. They point out that one fact is brought out with certainty, the course of motor fasciculi is a straight one from the point where the nerve has been made up by its contributing segments to the point of offset of the fasciculi as a branch. This work permits conclusions opposed to those of Dustin,[20] who called attention to the presence of intraneural plexuses between each branch of a nerve, and of Langley and Hashimoto.[21]

Our knowledge of experimental regeneration of nerves has been enriched by the American investigators, Lewis and Kirk [22] and Huber.[23] It is definitely established that regeneration of a severed nerve occurs from the outgrowth of the axons of the proximal segment. The work of Huber on direct suture and various forms of transplants, fresh and preserved, has indicated the proper surgical treatment of various peripheral nerve lesions. Lateral implantation and nerve plastics have been discarded. The surgical treatment of these injuries seems to offer a better prognosis in the hands of American than other operators. Valuable contributions on surgical treatment have already

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15. Roussy and Lhermitte: Military Medical Manual, The Psychoneuroses of War. Author: Please give publisher and year.

16. Babinski: Rev. Neurol., April-May, 1916.

17. Marie, Meige and Gosset: Compt. rend. Acade. d. méd., Dec. 28, 1915.

18. Déjerine, J. J., and Déjerine, Mme.: Presse méd., No. 13 (March 2) 1916.

19. Kraus, W. M., and Ingham, S. D.: Electrical Stimulation of Peripheral Nerves Exposed at Operation, J. A. M. A. **74**:586 (Feb. 28) 1920.

20. Dustin: L'Ambulance de L'Ocean, 1917.

21. Langley and Hashimoto: Quoted by Kraus and Ingham.

22. Lewis and Kirk: Bull. Johns Hopkins Hosp., February, 1917, p. 71.

23. Huber: Arch. Neurol. & Psychiat., 1919. Author: Please give volume and page.

appeared by Stookey,[24] Dean Lewis,[25] Frazier,[26] Spear and Babcock,[27] Price Feiss and Terhune [28] and Elsberg.[29] Although sufficient time has not elapsed for definite conclusions to be drawn, a promising outlook is afforded by their reports. L. J. P.

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24. Stookey: Surg., Gynec. & Obstet. **23**:639 (Dec.) 1916.
25. Lewis: Surg., Gynec. & Obstet. **30**:528 (May) 1920.
26. Frazier: Ann. Surg. **71**:1 (Jan.) 1920.
27. Spear and Babcock: Arch. Neurol. & Psychiat. **2**:253 (Sept.) 1919.
28. Price, Feiss and Terhune: Arch. Neurol. & Psychiat. **1**:547 (May) 1919.
29. Elsberg, C. A.: Technic of Nerve Suture and Nerve Grafting, J. A. M. A., **73**:1422 (Nov. 8) 1919.

Abstracts from Current Literature

PHYSIOLOGISCH-ANATOMISCHE UNTERSUCHUNGEN UEBER DIE SOGENANNTE ARMREGION DER GROSSHIRNRINDE (PHYSIOLOGIC-ANATOMIC INVESTIGATIONS OF THE SO-CALLED ARM REGION OF THE CORTEX). Hans Meier-Mueller, Schweiz. Arch. f. Neurol. u. Psychiat. 5:270, 1919.

In this article Meier-Mueller reports the grosser pathologic and histopathologic findings of the brain of a chimpanzee (*Troglodytes niger*), "Billy," who was operated on by Graham Brown and Sherrington. They removed the arm region of both sides in six successive operations, three on the left and three on the right, in the course of about sixteen months. The arm centers had been located by electrical stimulation at the time of each operation; following the operation there was in every instance a total paralysis of the opposite arm, which recovered with very "little change" in movements ultimately. The animal died nine days after the sixth operation.

A preliminary report of these investigations was made by Graham Brown and Sherrington at the Ninth International Congress of Physiologists in Groeningen in September, 1913. Monakow, who was present at this time, noted that definite synkinesias were present in the movements of the arms, although the animal was able to perform such movements as pouring water into a glass and drinking it. The brain was sent to Monakow's institute, where its examination was undertaken.

The fact that no permanent paralysis could be produced necessitated revision in our views concerning the function of the so-called motor area of the cortex. Grossly, the defect produced by the operations on the right hemisphere included destruction of the subcortical portion of the second frontal convolution, from its middle portion, in an anteroposterior direction and extending upward to the sulcus of the first frontal gyrus, backward through the field of operation, and including the corresponding portion of the postcentral gyrus; the flocculus, centroparietal fasciculus and deeper structures of the brain were not involved. On the left side the defect extended in a fronto-occipital direction from the middle third of the first frontal convolution, including its mesial edge, through the posterior two thirds of the second frontal, in which half the cortex of the sulci remained intact, through the so-called arm center of the anterior central gyrus, into a small portion of the postcentral gyrus lying opposite. The defect, therefore, on both sides included those portions of the arm centers which could be excited electrically, located in the middle third of the precentral gyrus, the caudal third of the second frontal, and a portion of the postcentral, particularly on the right, including the greater portion of the corresponding medullary substance, without involvement however of the centroparietal fasciculus or the pyramidal radiations. The neighboring "leg center" and "head center" were not involved. Examination of the thalamic region revealed an extensive degeneration of the middle segment of the optic thalamus in a fronto-occipital direction, slight changes in the caudal portion of the frontal third, as well as in the dorsolateral portion of the lateral nucleus; the medial portion of the nucleus ventralis b. was more or less extensively involved.

While the thalamic representation of the cortex is now fairly well understood, this furnished an exceptional opportunity of delimiting more sharply that

portion of the thalamus corresponding to the arm region. It was noted, in a general way, that on both sides degenerative changes occurred in the thalamus in the caudal portion of the frontal third and in the entire middle third in the anterocaudal direction, in the lateral portion, of the middle third, in a dorsoventral direction; on the right side, there was in addition degeneration of the medial portion of the nucleus ventralis b. The arm region, therefore, would roughly correspond to the middle third of the lateral nucleus of the thalamus. The portion of the postcentral gyrus, destroyed only on the right, would correspond to the medial third of the nucleus ventralis b.

In animals operated by Munk, in which the precentral gyrus had been completely removed, the entire lateral nucleus, in a dorsoventral direction, was degenerated. The writer infers that the cortical representation of the frontal third of the thalamus must be the frontal convolutions. Monakow is of the opinion that the tuberculum anterius corresponds to the medial portion of the first frontal convolution and the paracentral area.

From the physiologic standpoint, movement comprises exceedingly varied and complex factors. Monakow noted certain synkinesias in the arm movement of this monkey. Consequently some of the isolated synergistic movements must have been hampered. This observation appears to refute the generally accepted view that the so-called arm region alone contains the anatomic and dynamic substratum of the manifested "kinetic melodies." Only certain types of movements requiring a fine reciprocal play between agonists and antagonists, or, as Monakow puts it, the ability for reciprocal inhibition of the extraneous and interfering movements of the spinal and subcortical synergies arising in a reflex manner, call for the integrity of the "foci aggregates" of the arm region.

The author next cites three clinical cases which he thinks support this assumption:

The first case was that of a man 21 years of age, whose head had been injured by a wagon wheel three years previously, following which he developed jacksonian attacks of the right arm, which became paralyzed. At operation an encephalitic area was completely extirpated. Following operation, the patient suffered a right hemiplegia, an aphasia and a psychic disturbance, characterized by dulness and disorientation. His condition improved, however, so that eight years later he was graduated from school with an excellent record. The only evident symptom which persisted was a fine tremor in the right upper extremity. Isolated movements of the fingers were equally good in both hands.

The second case was that of a 35 year old farmer's wife, who had jacksonian attacks of the right arm. At operation an oval tumor about 3 cm. long was excised. The right arm was completely paralyzed. Four months later the patient was able to perform isolated movements of the fingers, which, however, were somewhat slow and awkward.

The third case was that of a woman 40 years of age suffering from jacksonian seizures of the right arm, an accompanying right-sided hemiparesis, projectile vomiting and choked disk. An encapsulated abscess was removed from the left precentral gyrus, following which there was a complete right-sided hemiplegia with aphasia. One month later the patient was able to use her hand in dressing and in her housework—however, with a little weakness, slowness and awkwardness.

Every movement is composed, philogenetically, of old and recent, as well as static-dynamic and orientation components. Elementary movements occur in the fetus; these develop into the locomotor reflexes, the rhythmic, orienting, purposeful and expressive movements of the adult. Their localization must

necessarily be complex and not delimitable to a given point. It is quite certain that the division of labor necessitates numerous "centers" for given functions, some of which remain subcortical, while others wander to the cortex and toward the frontal area, depending on the philogenetic position of the animal. Just as movement becomes progressively better developed from earliest childhood, so the anatomic localization becomes correspondingly more complicated and diffuse, and doubtless comprises, anatomically, centers that cannot be excited electrically. The author feels that the designation "center" should be replaced by that of "region."

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REVIEW OF EIGHT YEARS' EXPERIENCE WITH BRAIN TUMORS. ERNEST SACHS, Arch. Surg. **1**:74 (July) 1920.

Sachs, in this article, gives some of the results of his experience in operating on a series of eighty-five cases of brain tumor. His experience is valuable because he is devoting his entire time to neurologic surgery, one of the very few to do so in this country. As he states, Cushing is quite right in saying that the time has passed when there is any purpose in speaking of the mortality in brain tumor cases any more than there is in speaking of the mortality in abdominal tumor cases. Too often we find pessimism even in the ranks of neurologists. The mortality is admittedly higher than in other fields of surgery, but often operation offers the only chance of relief. Sachs is frank in stating his own mortality—35.5 per cent.; but, as he points out, we find the predominating mortality—eighteen out of twenty-nine—in glioma cases, in which type of tumor we have been accustomed to think of the growth as usually inoperable from the standpoint of removal. Sachs successfully removed 14 per cent. of the gliomas in a series of twenty-one cases; a creditable record. He points out that those gliomas that grow by replacing tissue do not give rise to any general symptoms of increased pressure and we can therefore only depend on local phenomena irritative or paralytic in character. It serves a useful purpose to call attention to the fact that such phenomena may be caused by a tumor which, if operated early enough, may be removable. Sachs states that in the cases of glioma successfully removed symptoms were only present for four months or less and that four weeks should be the longest time to try palliative measures. There is little danger in an exploratory craniotomy in the hands of one trained in neurologic surgery and there is no doubt that such surgeons should be called on oftener to do these operations; that, in fact, such a procedure should be the rule when a brain tumor is suspected and the symptoms have not abated after a short trial of palliative procedures. The reviewer has in mind, of course, only those cases in which surgical removal offers a possible chance of cure and admits that a large number of tumors are so situated as to make surgical removal impossible. Even in such cases decompression may offer great relief.

Sachs calls attention to the value of ventricular puncture on the opposite side from the tumor as well as a preliminary decompression to relieve intradural pressure. There were sixty-four cases presenting tumors other than true brain tumors. In this group there were eleven deaths, a mortality of 17 per cent. Twelve of the cases were cysts. In thirty of the sixty-four cases there were no localizing signs and a decompression was performed to relieve general pressure symptoms. Sachs states that palliative procedures often afford a surprising amount of relief and for a considerable time, a fact with which the

reviewer has been impressed. Although the number of cases reported in this paper is not large when viewed from the standpoint of a statistical study, the group is sufficiently large to give results encouraging to their reporter and to those of us interested in neurologic surgery.

RODMAN, Philadelphia.

ESSAI D'APPLICATION DES TECHNIQUES DE LA PSYCHO-PHYSIOLOGIE ET DE LA PSYCHOLOGIE EXPERIMENTALE A LA NÉURO-PSYCHIATRIE (AN ATTEMPT TO APPLY THE TECHNIC OF PSYCHOPHYSIOLOGY AND EXPERIMENTAL PSYCHOLOGY TO NEUROPSYCHIATRY). RAOUL MOURGUE, Schweiz. Arch. f. Neurol. u. Psychiat. **5**:240, 1919.

The first part of this study on "The Psychomotor Function of Inhibition Studied in a Case of Huntington's Chorea," was abstracted in the ARCHIVES OF NEUROLOGY AND PSYCHIATRY **3**:663 (June) 1920. The author continued the study by investigating the reaction time of the patient to sensory excitations. He had already reported the study of simple reactions, auditory and visual, and of the time of reaction of choice with inhibition. Many of these experiments were conducted under great difficulty, since those ordinarily in use are applied to persons having normal attention and presuppose a certain degree of integrity of intellectual autoconduction. This is far from being the case with the subject of this article, and the precision of these experiments is apparent rather than real.

In the next experiment the patient, having his eyes bandaged, was asked to respond to the blow of a hammer on the table following the command of "attention." The same experiment had been carried on in August, when it was noted that the mean time of response was 0.25 of a second, that for the normal, being, according to Warren, from 0.12 to 0.23 of a second for auditory sensations. One month later, under exactly the same conditions, the reaction time was extended to 0.36 of a second, which is quite different from that first noted. The inconstancy of the various figures obtained is interpreted by the writer as an indication of pathologic distraction.

The time of reaction for simple visual stimuli, employing Pluecker's tube, giving a red light, was 0.71 of a second, which represents a very much higher figure than Warren gives for normal, which is 0.17 to 0.29 of a second.

In testing the time of reaction with choice Mourgue used the modification of Tolouse. It consists of a double handle of copper, which the subject is commanded to move from within out, sometimes with the right hand, at other times with the left, depending on the intensity of the stimulus noted, for the purpose of arresting the hand of a clock, which was set in motion synchronously with the giving of the stimulus. A mean of 1.29 seconds was obtained. The time of discrimination was, therefore, considerably extended—however, discrimination was not abolished since the patient made but two mistakes, one of which he corrected later.

The time of visual reaction with complex choice was obtained by asking the patient to move the copper handle to the right with the right hand on seeing a red light, to the left with the left hand on seeing a white light, and to desist from movement entirely on seeing a green light. Errors in this test were frequent.

A test of perception was performed by asking the patient to name twenty-four objects with which he was familiar; three of these were named inac-

curately and one, which was a piece of chalk, was not recognized at all. The time for doing this was 2 minutes and 40 seconds, while the normal time is given as 20 to 25 seconds.

Marie and Lhermitte look on chronic chorea as a disease of both constitutional and acquired origin; constitutional in so far as it represents a congenital lack of resistance of the brain, and acquired through endogenous or exogenous intoxication, which starts off the degenerative process in the corticostriate region. The metabolism of these subjects has been little studied. Pighini and Juan Alzina y Melis have found that there is increased production of creatin and lactic acid, bringing about an increased creatinin and ammonia output in the urine, which, as they remarked, is probably a phenomenon secondary to the abnormal activity of the neuromuscular system. Probably the same explanation attaches to the absence of lipocholesterol inclusions noted in the suprarenals. In view of the paucity of these investigations, the writer feels that his finding of an increased albumin and lymphocytic content of the spinal fluid, indicating a chronic meningo-encephalitis, is of great interest.

Clinical observation of this patient leads the writer to interpret the state of dementia, according to the classic idea, as essentially one of confusion. Synthesis of the psychologic phenomena, in the presence of motor disturbances, demands further attention for, as Maudsley says, "who cannot govern his muscles, cannot govern his attention." Intellectual enfeeblement, which is not accompanied by distorted ideas, demands a minute and methodic investigation heretofore unpracticed.

The motor theory of psychic phenomena gives a satisfactory explanation of the intellectual and affective phenomena (loss of attention and irritability) here noted, which long have attracted the attention of clinicians. It alone permits a correlation of the anatomic and pathologic findings of Huntington's chorea, as described by Pierre Marie and Lhermitte, of the clinical observations, and of the findings obtained by psychologic experimental methods. The function of psychomotor inhibition seems, from a morphologic point of view, to be related to the neokinetic system of Ramsey Hunt. This is a rational application of the theory of "chronologic localization," expounded by Monakow, on which depends the future neuropsychiatry.

WOLTMAN, Rochester, Minn.

PRINCIPLES OF PERIPHERAL NERVE SURGERY. DEAN LEWIS, J. A. M. A. **75**:73 (July 10) 1920.

The author reviews briefly the principles involved in the process of degeneration and regeneration of peripheral nerves, emphasizing conditions that hinder and assist regeneration following neurolysis or nerve anastomosis. Scar tissue forms rapidly after the division of a peripheral nerve, and the presence of a neuroma may be expected within nineteen days. Since the neuraxes must enter the distal segment in the process of regeneration, a neuroma naturally interferes with its progress and should be resected to a point at which healthy funiculi appear and no scar tissue is visible to the naked eye. It is important to preserve the nerve pattern in the anastomosis, which should be performed as early as possible after the injury, about three and one-half months, but not until all evidence of infection has disappeared. Results at this time will be better than can be obtained six months later. The author states emphatically that "end-to-end suture is the only procedure that

can be relied on to reestablish the continuity and function of a nerve after division." It is immaterial whether silk or catgut is used in the anastomosis, and a covering is not necessary. Autogenous transplants and fascial tubulization prove successful in dogs, but not in man.

One hundred and twenty operations are reported; in twelve of these autocable transplants were used and in six tubulization with fascia. In none has there been recovery, although in two transplantations there has been a slight return of motor power in isolated muscles. Tinel has stated that neurolysis is permissible only when it restores a free, supple nerve in the interior of which is found no obstacle to regeneration and that, as a whole, liberation is ineffective in cases of severe lesion of nerve trunks associated with rupture of the sheath, and the development of scar tissue with the formation of a neuroma. Neurolysis is of distinct value, but it is sometimes difficult to determine whether or not the results are the direct results of surgical intervention; nerves presenting the most marked pathologic changes have recovered spontaneously.

Whether neurolysis or resection is to be performed depends on the appearance and feel of the nerve and the electrical reaction. It should be combined with capsulectomy, that is, removal of the thickened epineurium, or parallel incision to it, and transference of the nerve between normal muscle fibers away from the bed of scar tissue. The musculocutaneous nerve is the first to regenerate, then the musculospiral, the internal popliteal, and the median. The external popliteal is the slowest to recover.

Aside from the operative procedure, the author emphasizes the importance of postoperative care, the use of massage, and measures to prevent overstretching of muscles; he also calls attention to the fact that fibrous changes in a muscle or ankylosed joint defeat the object of peripheral nerve repair.

The article is especially valuable to the clinician in that it states briefly the process of degeneration and regeneration, emphasizes the surgical indications, and shows the comparative value of end-to-end anastomosis with that of fascia tubulization and autogenous transplants.

ADSON, Rochester, Minn.

LE MONGOLISME (MONGOLIANISM). E. APERT, Bull. méd. **34**:563 (June 19) 1920.

In the extremely varied group of abnormal infants, the author considers congenital myxedema and mongolianism as two special groups, each different from idiocy, imbecility, retardation of evolution or mental anomaly based on congenital lesions or on defects of cerebral development. His paper deals with the second of these groups. A large portion dealing with the clinical features may be passed over as familiar. Regarding teeth in these cases: The eruption of teeth is delayed. The first teeth sometimes do not come until the tenth, fifteenth, or twentieth month. The order of their appearance is irregular and quite often certain teeth are lacking, particularly the lateral incisors. The molars appear late, being delayed until the third, fourth or even fifth year. The second dentition is retarded and irregular, but one does not find in general, the microdontism, the dysmorphisms, or erosions of enamel seen in hereditary syphilis. The dental fragility and early caries seen in myxedema are also absent.

In the great majority of cases seen by the writer he has been able to exclude syphilis as an etiology. He has found that the mongolian is frequently

the last of a numerous family, the mother relatively advanced in age and weakened by frequent pregnancies. Often he has found that in the months of pregnancy mental and moral factors, distressing to the mother, were present.

He believes that he can benefit cases of mongolianism by opotherapy, using thyroid and suprarenal glands. He writes: "I use with the powdered organs manganese, which appears to be the metal-ferment of the suprarenal gland, and chalk and magnesium, which appear to be the metal-ferments of thyroid and parathyroid." For an infant in the first year he gives at the beginning:

	Cg.
Suprarenal (whole gland), dried and powdered	0.02
Thyroid, dried and powdered	0.01
Bioxid of manganese	0.01
Glycerophosphate of chalk	0.05
Glycerophosphate of magnesium	0.05
Sugar, powdered	0.30

This is used for four days continuously and then omitted for three days. The dosage is gradually increased every second week or so, and even as much as 10 cg. of suprarenal gland by day can be given an infant of 1 year.

After outlining the features of improvement that may be expected, he writes, "the mongolians will always need guidance. They will lack initiation and judgment. . . . They will remain always below standard but can be sufficiently restored so as to escape the special asylums where until recently they were confounded with the true idiots."

DAVIS, New York.

FINGER EXPLORATION OF GUNSHOT WOUNDS OF THE BRAIN.

EDWARD B. TOWNE and T. R. GOETHALS, Ann. Surg. **71**:531 (May) 1920.

The authors emphasize the value of finger palpation of gunshot wounds of the brain and state that foreign bodies are frequently overlooked by both curettage and catheter palpation. They report a series of sixty-one of the head wounds operated on by the surgical team at the casualty clearing stations of the British First Army from May 12 to Aug. 12, 1918. Eleven of the wounds were simple scalp wounds, and twenty-two were fractures with unopened dura; none of these thirty-three cases brought out data of particular interest. Their discussion is confined to twenty-eight cases of skull fracture associated with lacerations of the dura and brain. Twenty patients were operated on; eight were inoperable. The cases are reported in detail, and attention is called to the fact that thirteen of the patients evacuated from the clearing station were followed for periods of from six months to twenty-one months after operation.

The authors give an excellent review of the literature on the treatment of various types of skull injuries. They outline the advantages and disadvantages of the four principal methods employed in the treatment of severe brain wounds, namely, (*a*) extraction of metal with the magnet or under the screen, (*b*) gentle curettage of the cavity, (*c*) catheter palpation with forceps extraction, and (*d*) finger palpation with forceps removal of the foreign body. The method best adapted to the individual case should be followed, bearing in mind the fact that finger palpation is indispensible when the wound is sufficiently large to admit the finger. Since the brain wound frequently is much larger than the dural opening, the dural wound should, if necessary, be opened.

The routine treatment instituted by the authors was briefly as follows: Unless obviously a subject for general anesthesia, local anesthesia of procain was employed. The damaged scalp and pericranium were removed, the wound treated with tincture of iodin, and the involved skull removed in block. A 17 F. soft rubber catheter with an attached syringe was used to aspirate the necrotic cavity and, if the defect in the dura was large enough, a finger was introduced gently and the foreign bodies removed with forceps. If the defect in the dura was too small to admit a finger but information pointed to a cavity in the brain large enough for finger exploration, the dura was incised to allow the finger to enter. The wound was irrigated with saline, hemorrhages were controlled, and the dura was sutured partially or, if possible, completely with fine chromicized catgut or silk. A few plain catgut stitches were used in the galea and silkworm gut in the skin. A small gutta percha tissue wick was introduced to the bone if hemostasis was doubtful. During the convalescent period, the wound was inspected daily; if there was any evidence of tension or presence of headache, a spinal puncture was made and repeated as often as indicated. The sutures and wick were usually removed in from twenty-four to forty-eight hours.

The article is timely in that it reviews extensively the literature on the treatment of skull and brain wounds and emphasizes practical surgical procedures.

Adson, Minnesota.

LE SYNDROME PARKINSONIEN DANS L'ENCEPHALITIS LETHARGIQUE (THE PARKINSON SYNDROME IN EPIDEMIC ENCEPHALITIS). Pierre Marie in collaboration with G. Levy, Bull. de l'Acad. de méd. **83**:539 (June 15) 1920.

The writer concerns himself with points of difference between the postencephalitic parkinsonian syndrome and paralysis agitans (true Parkinson's disease).

In the beginning he points out that the lesion of paralysis agitans is considered by some to be the central nuclei, particularly the lenticular nucleus, while Tretiakoff believes the lesion is in the cerebral peduncles, especially in the region of the locus niger.

The age at onset is outstandingly unlike, generally speaking. Paralysis agitans rarely begins before 45. The parkinsonian syndrome following epidemic encephalitis occurs most often between the ages of 20 and 40. He has seen a case of epidemic encephalitis showing the parkinsonian syndrome in a child of 12.

Secondly, the preexistence of a general affection, usually febrile, can be found in all the instances of the postencephalitic condition. Headache, vertigo, diverse pains more or less localized, and ocular troubles are features not found in paralysis agitans.

The mode of onset is very different. In paralysis agitans the phenomena progress slowly. The other member on the same side of the body also becomes involved several months following the involvement of the initial extremity. Because of this sequence, the writer remarks that in paralysis agitans the mode of onset is "progressively hemiplegic." In sharp contract to all this, the onset of the parkinsonian syndrome in encephalitis is abrupt and also the extremities as well as the trunk are involved en masse, at the same time. The progressive hemiplegic mode is altogether lacking.

Fourthly, the writer has yet to find a postencephalitic case showing the tremor which is typical of paralysis agitans—the minute tremor which is temporarily in abeyance at the moment of voluntary movement. In postencephalitic cases the tremors are of large amplitude and are either released or augmented by voluntary movement. Spasmodic jerking movements of the face are seen in postencephalitic cases and not in paralysis agitans.

Abnormalities in the use of the tongue, in the opening and closing of the mouth and in mastication, present in some of the postencephalitic cases, are absent in paralysis agitans. That these particular differences arise is not surprising in view of the frequency in epidemic encephalitis of lesions of the peduncular-bulbar trunk.

Lastly, there is the difference of evolution. In the postencephalitic condition the syndrome has often almost completely cleared up, while in true paralysis agitans neither a cure nor even a recession of the symptoms is possible.

DAVIS, New York.

HAND AND FOOT PRINTS AS RECORDS OF THE PERIPHERAL NERVES. LEWIS J. POLLOCK, J. A. M. A. **74**:943 (April 3) 1920.

Graphic methods of recording signs and symptoms have in many instances a greater value than descriptive methods. It is frequently impossible to get photographic records of hands and feet in cases of peripheral nerve lesions; it is then serviceable to record the contour of the palm and that of the sole by the aid of impressions. Imprints show distinctive changes in lesions of five of the peripheral nerves, namely, the ulnar, the median, the radial, the internal popliteal and the sciatic, and also in combined lesions of the ulnar and the median.

Imprints of the hand in cases of ulnar nerve lesions show clawing of the inner two fingers, atrophy of the hypothenar muscles, nonseparation of fingers and extension of the first phalanx of the thumb. Atrophy of the adductor pollicis is demonstrable from the imprint.

Median nerve lesions show clearly the disturbance of the whorl formation on the tips of the index and middle fingers. When severe clawing is present in these two fingers, it is made evident by the absence of any imprint of the central portion of the palm. The failure of desquamation and the presence of many new lines over the thenar eminence are demonstrated.

Imprints of radial nerve lesions are characterized by the cramped appearance of the fingers which results from the inability to place the palm in a flattened position on the paper. The most characteristic feature of this imprint is the position of the thumb, which is adducted, the distal phalanx falling within or on the border of the outline of the index finger. The thumb is rotated about its own axis inwardly; and its distal phalanx is usually flexed. Absence of the signs of atrophy of the thenar and hypothenar eminences is an additional feature of this form of lesion.

In combined lesions of the ulnar and median nerves, signs of atrophy of both the thenar and the hypothenar eminences are demonstrable in the impressions by means of the notches found along their borders. Clawing is present in all four fingers.

Lesions of the sciatic nerve show, besides a slight pes cavus in some cases, a clawing of the toes indicated by the absence of their imprint on the paper. It may be stated that, although by no means diagnostic, such records are of

considerable value in determining the progress of the condition of atrophy and deformity in peripheral nerve lesions. The imprints are easy to take and require a minimum of time for the amount of record produced.

OTT, Rochester.

REPORT OF THE CONDITION OF A CASE OF COMPLETE DIVISION OF THE SPINAL CORD BY GUNSHOT WOUND NINETEEN YEARS AFTER THE INJURY. WILLIAM B. CADWALADER, Ann. Surg. **71**:719 (June) 1920.

The author describes a case which was reported by Drs. Francis T. Stewart and Richard H. Harte in the *Philadelphia Medical Journal* of June 7, 1902, under the title of "A Case of Severed Spinal Cord in which Myelorrhaphy was followed by Partial Return of Function."

The patient in question was shot in the back with a 32 caliber pistol, the bullet entering 1 inch to the right of the seventh dorsal spine and passing directly into the spinal canal. There was immediate and complete motor and sensory paralysis below the level of the tenth dorsal spine, and all the superficial and deep reflexes were abolished. Three hours after the injury an operation was performed by Stewart. The laminae of the seventh and eighth dorsal vertebrae were removed; the membranes were found to be lacerated, and the spinal cord was completely severed. There was a distance of three-fourths inch between the severed ends of the cord. The divided ends of the spinal cord were approximated with catgut sutures and the wound closed. Surgical recovery was uneventful.

The patient was examined repeatedly by different physicians and was exhibited before many medical societies with the impression that there was some return of function. In making an examination recently, the author reports a complete paralysis of motion and sensation below the level of the tenth thoracic spine. The lower limbs feel rigid, but there is no wasting. The skin of the legs is somewhat waxy in appearance and pits slightly on pressure. The defense reflexes have developed, with the development of an automatic control of the bladder. The author states that the occurrence of any return of function must emphatically be denied; that such a statement was purely the result of error in making the observations.

The article is especially valuable in that it emphasizes the importance of a thorough neurologic examination, and substantiates the fact that regeneration cannot occur following myelorrhaphy. It is possible to occur only when the lesion is in the cauda equina, and one is able to anastomose the nerve roots after they have emerged from the cord substance.

ADSON, Minnesota.

ON THE RELATIVE VASCULARITY OF VARIOUS PARTS OF THE CENTRAL NERVOUS SYSTEM OF THE ALBINO RAT. EDWARD HORNE CRAIGIE, Jour. Comp. Neur. **31**:429 (June 15) 1920.

The author has made the first exact quantitative comparison of the vascularity of different parts of the central nervous system. Measurements of the richness of capillary supply were made in twenty-one regions of the spinal cord, medulla oblongata and cerebellum, and the results are presented in the form of tables and graphs.

The gray matter is more richly vascularized than the white matter, the poorest part of the gray being nearly half as rich again as the richest part of

the white. All parts of the white matter are not equally vascular, the pyramidal tract, the richest part in the spinal cord, being about twice as rich as the fasciculus cuneatus, while the fasciculus longitudinalis dorsalis in the medulla is still richer.

The gray centers can be sharply divided into two groups, the motor nuclei and the sensory and correlation centers, of which the latter are richer than the former. Though the richest motor region (ventral cornu) is but little poorer than the poorest sensory one (the fifth spinal nucleus), the two groups do not overlap in the case of those regions studied, except in a few individuals. The substantia gelatinosa Rolandi of the spinal cord is the only part which does not conform with this statement. The richest of the centers observed is the dorsal cochlear nucleus, which is more than half as rich again as the ventral cornu, about two and one-half times as rich as the substantia gelatinosa Rolandi (the poorest gray region), and eight times as rich as the fasciculus cuneatus. Great individual variations occur, and the two sexes do not seem to show any constant difference.

The interpretation of the functional significance of the anatomic data recorded depends on the unsolved problems of the fundamental nature of nervous processes and their relation to metabolic activity. The bearing of various theories regarding these on the present investigation is discussed.

HERRICK, Chicago.

RECOVERY FROM TUBERCULOUS MENINGITIS AFTER TREATMENT WITH INTRASPINAL INJECTIONS OF ANTIMENINGOCOCCIC SERUM. A. W. HOLLIS and T. H. PARDEE, Arch. Int. Med. **26**:49 (July) 1920.

Despite the comparative frequency of tuberculous meningitis, the fact that the literature contains only thirty-eight cases in which there has been recovery shows the high mortality of the disease. The authors have used intraspinal injections of antimeningococcic serum as a new therapy in tuberculous meningitis. This has been used by only one previous worker (S. Schaeffer, 1913) and then in a single case. In their series of seven cases the authors report recovery in four. Of these four, the diagnosis in two cases was confirmed by demonstration of tubercle bacilli in the spinal fluid; in the other two cases convincing clinical proof for the diagnosis is offered. Concise case reports are given.

Removal of the spinal fluid always preceded the injection of serum. The amount of serum injected was 20 c.c. or somewhat less, and the first two or three injections were given at daily intervals and then every second or third day. No patient received more than five injections. The cases with demonstrated tubercle bacilli in the spinal fluid received four and five injections, respectively, and recovered. The development of serum sickness appeared to hasten the therapeutic response.

Concerning the manner of this response, theories are briefly considered. The writers believe that "the intraspinal injection of antimeningococcic serum (in cases of tuberculous meningitis) has two distinct actions; first by adding to the spinal fluid certain antibodies which it is unable to develop itself and second, by the introduction within the dura of a foreign protein in the form of horse serum. The irritative effect of the latter on the meninges produces a cellular response and a hyperemia about the site of any localized tubercle with beneficial result."

This work is eminently important and should be widely noted in the hope that other clinicians may use the method and add to our knowledge of a promising new therapy.

DAVIS, New York.

DISCUSSION OF A CASE OF PARALYSIS OF BOTH PERINEAL NERVES. S. SLANSKY, Neurol. Centralbl. **39**: (Feb. 1) 1920.

The author reports a patient, 43 years of age, who was operated on for hernia, Oct. 8, 1918, under chloroform anesthesia. The thighs were strapped together just above the knee. On October 18, the patient experienced difficulty on attempting to walk, due to a double foot drop. November 11, examination revealed a bilateral peroneal paralysis, more severe on the right side. No other pathologic condition could be demonstrated. There was no sensory disturbance, as shown by direct and indirect faradic stimulation, although the contractions of the right leg were more sluggish. By galvanic stimulation no reaction of degeneration was elicited. The deep reflexes were reduced, but there were no pathologic sole reflexes or ataxia.

In his discussion, the author refers to the frequency of brachial plexus paralysis during operation. Brown states that this is accounted for by compression of the brachial plexus between the clavicle and the cervical vertebrae, due to severe abduction and extension of the upper arm. Attention has been given to the prevention of postoperative brachial palsies, but thus far the utmost care has not always proved successful.

In the author's experience this is the first instance of bilateral peroneal palsy occurring during operation and he questions whether or not the chloroform was the contributing factor and what influence the strap may have exerted. He states that moderate pressure over a considerable period might well be assumed as the cause, especially if forcible flexion or extension of the legs is permitted during the operative procedure. In this particular instance the paralysis was completely relieved after three weeks' treatment.

SHELDEN, Rochester, Minn.

AMPUTATION NEUROMAS: THEIR DEVELOPMENT AND PREVENTION. G. CARL HUBER AND DEAN LEWIS, Arch. Surg. **1**:85 (July) 1920.

The Great War has stimulated the study of peripheral nerve surgery enormously. It was the frequent occurrence of painful stumps in war amputations that led the authors to carry on this study in order to try to discover the cause of, and to prevent the development of, amputation neuromas. They state that as infection and hemorrhage are known to play an important rôle in the development of these neuromas that their experimental work was carried out with strict precaution to prevent both. In spite of the fact that they were entirely successful in this amputation neuromas invariably developed after experimental resection of the sciatic nerve in rabbits. Neuromas, as the authors point out, are due to the effort of the neuraxes to join the divided distal end. Being unable to do so the neuraxes are turned back on themselves by the connective tissue. The authors have shown by experimental work that the best way to prevent the development of such neuromas is to inject the nerve with absolute alcohol ¾ to 1 inch proximal to the proposed site of resection; if this is done there is an immediate nerve degeneration; the neuraxes in the injected areas are broken up into short irregular segments, myelin is present in the form of a granular detritus and smaller and larger globules. The neurilem-

mal sheaths appear well preserved. The longest time that elapsed from injection to removal of the nerve and microscopic studies was 175 days. By this time numerous neuraxes both myelinated and nonmyelinated had passed through the injected part of the nerve to the extreme distal end of the proximal segment. The connective tissue sheath of the nerve is materially thickened at this point. The structural appearances presented are not those of a neuroma. The use of alcoholic injection to cause nerve degeneration is a proved procedure, but its use to prevent the formation of neuromas is new and in the light of this report should be given a clinical trial.

RODMAN, Phildelphia.

APOPLEXY AND HYPERTONIA. ERNA HERRMANN, Neurol. Centralbl. **39**: (Feb. 1) 1920.

Strasser recently called attention to the fact that those with hypertension are subject to apoplexy only when the condition is associated with arteriosclerotic changes of the vessels or with chronic nephritis. Of ninety-seven hypertonics with arteriosclerosis, eight had severe apoplexy, and of twenty-nine nephritic cases, six were similarly affected. Thirty-four cases of simple hypertonia showed no severe apoplexy, although some had very high blood pressure.

The author studied forty-four cases of apoplexy to determine the relationship to arteriosclerosis, chronic Bright's disease and hypertonia. She calls attention to the difficulty of accurately distinguishing essential hypertonia from that based on organic disease, but she depends on the physical examination to determine this. The forty-four cases included twenty-two men and twenty-two women. One hundred and forty mm. of mercury were considered the upper limit of normal. Thirty-five of the cases showed a definite arteriosclerosis with symptoms dependent on this condition; three had an old valvular disease; four had chronic nephritis, and two had an old syphilis with positive Wassermann reaction. Of the forty-four cases none were found which could be classified as an essential hypertonia.

SHELDEN, Rochester, Minn.

CONGENITAL FACIAL PARALYSIS. FRANK R. FRY, J. A. M. A. **74**:1699 (June 19) 1920.

The author reports two cases of congenital facial paralysis of unusual interest. The first case, that of a child aged 1 month, was brought to the pediatric service of the outpatient department of Washington University School of Medicine with a complete right and a partial left facial paralysis. The second case was that of a young man, who gave a history of congenital weakness of the right facial muscles. In both instances the paralysis was more marked in the upper branches of the seventh nerve.

In the author's belief, congenital facial paralysis is due to a disease of the nuclei. Peripheral facial paralysis affects the whole side of the face, whereas in the central and in most of the bulbar types the superior branch retains its movement. In congenital cases the superior branch remains motionless, while the region about the mouth retains either complete or partial motility. If this difference between the upper and the lower part of the face is great, it gives a characteristic facial expression, such as full protruding lips, in contrast to the masklike, expressionless, sunken face mentioned above. The appearance of the face makes the condition often recognizable at the time of birth, and gives the impression of an advanced atrophy or aplasia of the muscles; also, the skin over the muscles is usually smooth, peculiarly shiny, and colorless.

ADSON, Rochester, Minn.

THE CRANIAL, OCCIPITAL AND ANTERIOR SPINAL NERVES OF THE DOGFISH, SQUALUS ACANTHIAS. H. W. Norris and Sally P. Hughes, J. Comp. Neurol. **31**:293 (June 15) 1920.

The dogfish, as a typical generalized vertebrate, has long been a classic subject for investigation of problems in vertebrate embryology, comparative anatomy and physiology. In recent years it has also been used extensively in medical schools as a dissection type to introduce the student to the general features of the anatomy of the central and peripheral nervous system and sense organs.

Norris and Hughes have given a detailed description of the peripheral nervous system in a paper of 110 pages with fifty-three illustrations. On the basis of serial microscopic sections of the entire head of young specimens, controlled by dissections of the adult, they have analyzed the nerve components, giving detailed and exact information regarding the entire peripheral courses of nerve fibers of all functional systems. Future students will have the benefit of this information regarding the central and peripheral connections of every functionally distinct kind of nerve fibers in the cranial nerve roots, ganglions and peripheral nerves. Comparisons are made throughout with the smooth dogfish, Mustelus.

Herrick, Chicago.

MEDIANUSLAEHMUNG MIT FOLGENDER PHRENICUSLAEHMUNG NACH SCHUSSVERLETZUNG (MEDIAN NERVE PARALYSIS FOLLOWED BY PARALYSIS OF THE PHRENIC AS THE RESULT OF GUNSHOT INJURY). Richard Meisner, Neurol. Centralbl. **39**:84 (Feb. 1) 1920.

On Oct. 29, 1918, a soldier, aged 23, was wounded 7 cm. below the coracoid process in the left arm, the injury producing permanent paralysis of the median nerve. The bullet emerged near the lower end of the left scapula. In January, 1919, there developed painful and restricted breathing and pain in the region of the stomach, especially after meals. Deep breathing and gaping produced pain in the left hand. The tenth, eleventh and twelfth dorsal vertebrae and ribs were painful on pressure. Also, 6 cm. above the middle of the clavicle pain was produced on pressure, which extended down the left arm. The left hand exhibited anesthesia, weakness and atrophy in the distribution of the median, and abnormal perspiration, cyanosis and glossiness. Roentgen-ray examination revealed a paralysis of the left diaphragm in a high and motionless position. This persisted until March, when its function became normal.

Two views are offered to explain the involvement of the phrenic nerve: First, by an extension of inflammation from the wounded area to the phrenic nerve by way of the lymphatics. Second, by an ascending neuritis along the median to the roots which are common to both nerves. The author favors the latter view.

Shelden, Rochester, Minn.

LETHARGIC ENCEPHALITIS IN PREGNANCY. Margaret Schulze, J. A. M. A. **74**:732 (March 13) 1920.

Lethargic encephalitis rarely occurs during pregnancy. Including the case reported by the author, there are only eight cases on record; of these, one patient recovered, five died, and in two instances the outcome was not reported.

The author reports the case of a woman, 35 years of age, one month past term, who died of pulmonary embolism on the twenty-sixth day after delivery. Microscopic examination of the brain revealed characteristic findings of lethargic encephalitis.

This disease appears to be far more common in the male than in the female—122 cases in males and 67 in females were reported.

OTT, Rochester.

THE OLFACTORY SENSE ORGANS OF ORTHOPTERA. N. E. McINDOO, J. Comp. Neurol. **31**:405 (June 15) 1920.

Dr. McIndoo in this and several preceding articles has contributed new observations on the olfactory organs of insects. Contrary to the prevailing belief, the antennae are not the chief organs of smell. This sense is served by "olfactory pores" widely distributed over the body surface. In the Orthoptera the total number of pores varies from 271 to 1,616.

HERRICK, Chicago.

LES MODALITES DE DEBUT DE L'ENCEPHALITE EPIDEMIQUE (TYPES OF ONSET IN EPIDEMIC ENCEPHALITIS). J. A. SICARD, Bull de l'Academie de méd. **83**:474 (May 18) 1920.

This brief paper concerns clinical types of epidemic encephalitis and emphasizes the occurrence of forms other than the oculolethargic originally described. For this reason, the writer deprecates the name lethargic encephalitis for the group.

It is in the early stages of the disease that the types stand out most distinctly—the oculo-lethargic, myoclonic, algic and paraplegic. The first type is already thoroughly recognized The myoclonic type often evolves totally free from lethargy or ocular troubles. Rhythmical twitching movements are present instead. These, when diaphragmatic in localization, are pathognomonic. The type with pain as an early feature—the so-called algic modality—can be confounded with many things—facial neuralgia, otitis, tabetic radiculitis, rheumatic neuritis, diaphragmatic pleurisy and renal colic. Diagnosis at the onset of this type is often impossible and must await the appearance of corroborative signs. The paraplegic type is more rare, though the writer has seen two cases. Here again diagnosis must be uncertain until the appearance of ocular troubles, lethargy or special muscular agitations point the way to the true situation.

DAVIS, New York.

Society Transactions

AMERICAN NEUROLOGICAL ASSOCIATION

Forty-Sixth Annual Meeting, June 1-3, 1920, New York

J. RAMSEY HUNT, M.D., *President*

STATIC AND KINETIC SYSTEMS OF MOTILITY. Presidential Address. DR. J. RAMSEY HUNT. This paper appears in full in this issue.

ACQUIRED DOUBLE ATHETOSIS. DR. WILLIAM G. SPILLER, Philadelphia. This paper appears in full in this issue.

DYSTONIA MUSCULORUM DEFORMANS. DR. E. W. TAYLOR, Boston. This paper appears in full in this issue.

BRADYKINETIC ANALYSIS OF SOMATIC MOTOR DISTURBANCES. DR. S. PHILIP GOODHEART and DR. FREDERICK TILNEY, New York. This paper will appear in an early issue.

DISCUSSION

DR. SMITH ELY JELLIFFE, New York, said that on hearing certain of the remarks concerning these papers he was reminded of some discussions among botanists in his student days between the so-called "splitters" and the so-called "lumpers." One group of botanists would go into the fields and bring back some twenty different species of violets; the other group would take these "species" of violets, place them all under one "species" and call the variants "sub-varieties." One is reminded of this by some of the suggestions made here: Oppenheim maintains that dystonia musculorum is a distinct disease, double athetosis is a distinct disease, dystonia musculorum with or without liver changes is a distinct disease, and Wilson's progressive lenticular degeneration is a distinct disease. All this is from the standpoint of the "splitters." This type of mind acts statically and pigeon-holes things into disease entities. Dr. Jelliffe believed that this entire group might best be regarded as a series of variants with general trends in one or another direction, according to the interruption of precise fiber tracts or synaptic junctions, which are located in or are passing to and from the striatum region. This region is one of extreme complexity, and not as simple as has been regarded by some of the speakers; to speak of the functions of the striatum, as though it were a simple structure, is naive. Of the various recent analyses of the striatum possibilities, which would have to include all of the complex pathways of this region, reference should be made particularly to the work of Cecile and Oskar Vogt,[1] who presented the best representation of the anatomy of this region. At a recent meeting of the New York Neurological Society, Dr. Abrahamson presented three cases of dystonia musculorum, which have been illustrated in Drs. Tilney

1. Vogt: Heidelberger Akad. d. Wissenschaften Biol., Series No. 14, 1919.

and Goodhart's moving picture analysis, and called attention to the opportunity for the study of the developmental phases of the dystonia musculorum group. Particular emphasis should be placed on the variety of clinical trends of a larger group of striatum syndromes of which these cases are merely striking types. It has become increasingly evident that dystonia musculorum, Vogt's double athetosis, Westphal's pseudosclerosis, Wilson's lenticular degeneration, Huntington's chorea, tuberous sclerotic idiocy (not mentioned by Dr. Spiller) and even paralysis agitans, are to be regarded as variants of the larger picture of striatum syndromes. One of Ziehen's patients came to necropsy and negative findings were reported, but in the more recent Flatau and Sterling case studies of the striatum and liver were made by Thomalla,[2] Schneider,[3] and von Economo.[4] This case presented a combination of the Ziehen-Oppenheim group of dystonia musculorum and Wilson's lenticular degeneration, since the clinical picture was characteristic of the former, while the hypertrophic sclerosis of the liver was typical of the latter. Abiotrophic atrophy of the cells of the putamen was found in the striatum. A synthesis of the various striatum syndromes, outlining the pathologic trends and enlarging the neurologic picture, has recently been attempted by the Vogts[5] and Bielschowsky.[6] The former[7] distinguish (1) the *état marbé* originally described by them as double athetosis (in this group Little's disease may be included); (2) *état dysmyélinique*, or progressive degeneration of the fibers passing between the striatum and the pallidum and the thalamus and hypothalamus (in one of Jelgersmas cases a paralysis agitans syndrome was present); (3) the *état fibreux*, progressing often as a bilateral progressive chorea without psychical symptoms; (4) an initially acute, then stationary status fibrosis as a partial expression of cerebral hemiatrophy (Bielschowsky) with nonprogressive athetosis and minimal paresis of body musculature; (5) total necrosis of the striatum confined chiefly to the putamen, at times reaching other parts of the globus pallidus (the dystonia case of Thomalla and Wilson's lenticular degeneration type both belong to this group); (6) acute infectious striatum syndromes (in our recent lethargic encephalitis epidemic many of these paralysis agitans-like cases, athetotic cases, pseudobulbar palsies, involuntary laughing and crying cases, etc., have been observed); (7) presenile degenerations involving chiefly the striatum and pallidum combined with neuroglia proliferations in striatum, and (8) status disintegratus with paralysis agitans-like symptoms, with the striatum involved either as (*a*) a general massive atrophy, chiefly of the caudatus, both ganglion cells and fibers, (*b*) small lacunar softenings or hemorrhages, and (*c*) rarefication and absorption, related to Marie's lacunar softening. The relationships of the striatum to the vegetative control of the muscle metabolism through the liver raises a point of speculative interest. Whereas Wilson argued that a toxin from perverted liver function caused the striatum changes, Dr. Jelliffe emphasized the neural integration disturbance of striatum interruption of vegetative liver pathways. Schneider's study offers several points of interest concerning the possibilities of an analysis of the vegetative integration mecha-

2. Thomalla: Ztschr. f. d. ges. Neurol. u Psychiat. **41**: 1918.
3. Schneider: Ztschr. f. d. ges Neurol. u. Psychiat.
4. Von Economo: Ztschr. f. d. ges. Neurol. u. Psychiat. **43**:173, 1918.
5. Vogt: J. f. Psychol. u. Neurol. **24**:1, 1918.
6. Bielschowsky: J. f. Psychol. u. Neurol. **24**:20, 1918.
7. Vogt: Zur Kenntniss der pathologischen Veränderungen des Striatum, Sitzsb. d. Heidelberg Akad. d. Wiss., 1919.

nism essential for liver function, interference with which causes vagatonic gliosis, to the disadvantage of the entire muscular metabolic functions, since it seriously impaired the integration of the involuntary part of the muscle structure, the sarcoplastic substance, and the sensorimotor, anisotropic disk parts. This latter speculative concept might have been raised by Dr. Hunt in his opening address.

Dr. Ernest Sachs, St. Louis, said that he had two observations which might have some bearing on the origin of athetosis. Some ten years ago he showed a girl with unilateral athetosis before the New York Neurological Society, and about a year later he removed her cortical arm center. About six weeks ago, he had a similar case. In both these instances the athetosis was absolutely stopped. Dr. Sachs said that he did not know exactly how to interpret this, but Monakow's theory might explain it. Sir Victor Horsley in 1908 published a case in which removal of the arm center for a similar condition also completely stopped the athetosis.

Dr. Frederick Tilney, New York, said that it seemed to him that from the pictures which have been seen, the classical terms for most of the defects due to hyperkinesis were inept and ineffectual for purposes of exact analysis of disturbed motility. If they are to be retained for future usefulness, they should be more fully defined. Chorea, for example, from χορεία, a dance, in itself has very little descriptive value. By connotation, it is associated with a large group of motor disorders that on finer analysis appear to have many fairly distinct characters, such, for example, as the flaccid chorea and the rigid chorea, the single muscle twitch, the muscle group twitch, and diffuse and associated muscle twitch. If chorea is a desirable term for these motor phenomena, then the several varieties must be carefully analyzed and grouped. The same is the case with athetosis, especially honored by American parentage, from the Greek ἄθετος, out of place, which is not descriptive of the motor phenomena indicated by it. Our conception of just what is meant by athetosis is not convincing. The invention of the hybrid choreo-athetosis emphasizes precisely how confused are our notions of this type of motor disorder. Generically, the conditions are probably closely related, but there are certain specific differences which must count heavily in the consideration of exact cerebral localization. Even dystonia musculorum deformans is not altogether free from this criticism, but requires much closer scrutiny and analysis. The danger inherent in all of these terms is that they offer an easy verbal escape from the necessity of careful thinking. The more convenient the terms are, the less apt are they to be exact. Our conceptions and definitions of the defects due to hyperkinesis require closer analysis before they assume real value in understanding the somatic motor organization and its defects. Hyperkinesis may disturb the attitudes and postures of rest by introducing purposeless and adventitious movements, it may deform the performance of volitional acts in the same way, and it may distort the performance of volitional acts and disturb the attitudes and postures of rest.

Dr. Charles K. Mills, Philadelphia, said that a decided advance has been made as regards the lenticular pathogenesis of cases similar to these brought brfore the society. The facts that were presented seemed to endorse more or less the belief that there is an association in the lenticular nucleus of impulses which are differentiated in the cortex. The remarks made by Dr. Sachs about the operations of Horsley and himself were possibly best interpreted along the lines of this theory. The choreic and athetoid movements, involuntary emotional expression, and various other phenomena which arise from destructive

lenticular diseases, in line with the principle enunciated by Dr. Jackson, are not active symptoms of the diseased part. Disease of the lenticular nucleus, because of the associations of this ganglion with the cortex, allows a freedom of movements necessarily aberrant stimulated by these cortically represented centers.

Dr. Gustave Roussy, Paris, France, said that he had the privilege of studying the extremely interesting case reported by Dr. Spiller in which the lesions were strictly limited to the putamen. The important thing in this case was the origin of the spasticity and contracture with increased reflexes and the Babinski sign, in the presence of complete integrity of the pyramidal tract at all levels. This finding was an apparent paradox if it is remembered that Dr. Babinski insisted, from the very start, that the reflex indicated organic or functional perturbation of the pyramidal tract. Several years ago, in studying thalamic lesion with his teacher, Professor Déjerine, the Babinski reflex was never observed in cases with lesions strictly confined to the thalamus.

Dr. Israel Strauss, New York, called attention to the moving pictures illustrating the cases of dystonia musculorum, in which in the hysteria case a most remarkable wavelike motion of the thigh occurred. In the last case of the dystonia group, the boy in whom it was said there was some improvement, a somewhat similar although not as intensive wavelike motion of the thigh was present. When he was admitted to Dr. Strauss' service at the Montefiore Home the diagnosis between dystonia and hysteria was not clear. When this patient bent forward, his back would curve normally, but when erect he had a marked lordosis. His arms were likewise affected. He improved greatly by reeducation but in the beginning his walk was very much worse. The curious dystonia movements of one arm also disappeared when he was sent to the workshop. The boy, strangely enough, could run very well. Dr. Strauss was still not in a position to say whether this case was one of true dystonia or hysteria, but he thought it worth while calling attention to the curious wavelike motion that was present. It is really a very important contribution of Dr. Tilney and Dr. Goodhart if this method of differentiation could be used in these cases, and if the wavelike motion could be utilized in differential diagnosis.

Dr. J. Ramsey Hunt, New York, said that in this discussion of the striatum he would recall briefly the interpretation of its function and symptomatology which he formulated in his study of paralysis agitans (*Brain*, 1917, p. 58) and which he thought clarified many of the difficulties of the situation. The corpus striatum contains two types of nerve cells: (1) A large motor cell which is found throughout the whole of the striatum, in the globus pallidus and also in the caudate nucleus and putamen of the globus pallidus type. This is the cell type which undergoes atrophy in paralysis agitans. (2) A smaller type of cells which are quite numerous in the neostriatum. These cells undergo atrophy in Huntington's chorea, the large globus pallidus cells being well preserved. There are, therefore, two fundamental cellular systems and two essential syndromes of the corpus striatum. (1) Chorea which is related to the neostriated system, and (2) paralysis agitans, which is related to the efferent pallidal system. When the large cells of the pallidal system are destroyed, the paralysis agitans syndrome results. If the small inhibitory and coordinating cells of the neostriatum are affected, there is chorea of the Huntington type.

There are other types of pathologic lesions which are more massive in character, and which may affect the two systems in different degrees, and that is the reason we have so varied a symptomatology in affections involving the

striatum. The symptomatology of Wilson's disease, dystonia musculorum, and double athetosis, simply represent degrees of involvement of these two essential systems.

Dr. William G. Spiller, Philadelphia, said he could not make any statement in regard to the Babinski reflex. The woman showed constant athetosis and the spastic condition was so intense that it was impossible to get sufficient relaxation to test this reflex.

Dr. E. W. Taylor, Boston, in response to a question as to whether the involuntary movements persisted during rest, said that it was noted in this as in other cases of similar character that these movements ceased entirely when the patient was sleeping or was not making muscular effort. The intention type of movement, if one may use that term, was very definitely defined. There were no electrical alterations and no muscular atrophy. In the moving pictures which Dr. Goodhart had shown the peculiar torsion movements which have been described were not evident. Unfortunately in the case which he himself presented, the slow moving pictures did not show the torsion movements but these were very evident in the rapid pictures. The patient in attempting to walk would often fall suddenly into a distorted position requiring him to proceed backward for a distance before he could again turn himself to face in the direction in which he desired to go. Mendel reported Thomalla's case as a doubtful one and Dr. Taylor was inclined to agree with him. The essential features were: marked involvement of face with disturbance in speech and deglutition; Babinski sign not always negative; associated athetoid movements. There were marked changes in the putamen, which was reduced in size on both sides and softened, but the nucleus caudatus and internal capsule were unaffected. The globus pallidus and the island of Reil were not involved. There was some enlargement of the thyroid, and the liver presented changes similar to those found in Wilson's disease and pseudosclerosis.

SUBTENTORIAL HERNIATION WITH HEMIANOPSIA. Dr. Adolf Meyer, Baltimore. This article appears in this issue.

A CLINICAL AND ANATOMIC STUDY OF A VASCULAR LESION LIMITED TO THE CEREBELLAR HEMISPHERES. Dr. Walter F. Schaller, San Francisco. This will appear in an early issue.

This paper discusses: marked asynergia, cerebellar catalepsy and scanning speech, absence of dysarthria, nystagmus, and of spontaneous errors of pointing after Bárány. It includes a demonstration of serial sections of the medulla, cerebellum, midbrain and basal ganglions by means of the epidiascope. It also discusses anatomic findings.

DISCUSSION

Dr. Charles K. Mills, Philadelphia, said that the case seemed very interesting in connection with the efforts recently made to determine the symptomatology and localization of destructive cerebellar lesions. Everything in the case pointed to the synergic function of the cerebellum, and there was nothing to indicate that the cerebellum is an organ directly connected with tonus. Catalepsy in cerebellar cases is rare and he had only seen one case, a man still living—in the wards of the Philadelphia General Hospital, who exhibited for a short time a sort of pseudocataleptic condition. This phenomenon of cerebellar catalepsy is not actually caused by any change in the cerebellum itself

but is due to the exercise of aberrant cerebral influence. The degenerations in the crus and other degenerations here shown in connection with a study of the symptoms, enforce the idea of the synergic function of the cerebellum.

Dr. LaSalle Archambault, Albany, said that the sections were beautifully stained and very well interpreted, but it seemed to him that it was extremely difficult to interpret the symptomatology observed in this case merely from the standpoint of the cerebellar lesions. Although the main lesions were found in the cerebellar hemispheres, there were a number of small foci throughout the brain stem, one in the pontine tegmentum, another presumably in the inferior olive, and a third in the crus cerebri. All of these lesions were small, and if they had been in the cerebral hemispheres it might not have meant so very much, but when located in the brain stem where so many nuclear complexes and fiber tracts are found, they might have a great deal of bearing on the incidence of the symptoms. Dr. Archambault did not believe that there was absolute justification in attributing the various symptoms to the foci in the cerebellum. The latter lesions, while not equal on the two sides, were symmetrical, and that may account for the fact that additional functional deficits were not found.

Dr. W. W. Schaller, in closing the discussion, said in answer to Dr. Mills, that this patient had no remarkable change of muscle tonus. He would designate cerebellar catalepsy in this case as a symptom of cerebellar disease without attempting to explain its exact mechanism. The objections of Dr. Archambault were explained on the basis of secondary degeneration occurring in the olive and the median fillet; the lesion in the median fillet and lateral fillet would hardly have any important bearing on the motor symptomatology of the case.

ELEMENTS OF PREDISPOSITION AND DETERMINING CAUSES OF SECONDARY DISORDERS IN PSYCHONEUROTIC ACROPARALYSIS RESULTING FROM THE WAR: THE DYSKINETIC SYNDROME. Dr. Gustave Roussy, Paris. This article appeared in full in the September issue, page 247.

An unusual syndrome with peculiar clinical manifestations was brought to light by the war. It is characterized by contractures resulting in fixation of the large articulations in defective postures, and by special motor difficulties (contraction, paralysis, or both) of the distal ends of the limbs. Some time following nervous shock or slight traumatism, impotency develops on a pithiatic basis and the immobility or defective function leads to organic accidents. This is a hystero-organic condition and must be considered in its elements: (1) the chief motor disorder, and the secondary (vasomotor, trophic, etc.) disorders. The motor disorder, the primary element, is of pithiatic character, capable of reproduction at the will of the patient and liable to disappear suddenly under favorable suggestion. The associated disorders, of variable character and very persistent, are comparable to admittedly hysteric disorders and are functional accidents based on predisposition to pithiatic motor disturbances. Investigation has shown that in predisposed patients there is marked bilateral vasoconstriction and microsphygmia; if hyperhidrosis or musculo-tendinous laxity is present this will also be apparent on the unaffected side, though less marked. Special predisposing associations lead to the production of articular disorders and of strong reflex reactions. Through psychic inertia, passive or forced immobility may aggravate preexisting tendencies,

and under the influence of a special neuropathic turn of mind and perverted will the persistent suppression of function causes the development of the secondary disorders. Restoration of activity leads to improvement.

DISCUSSION

Dr. Smith Ely Jelliffe, New York, said that the matter of extreme interest to him was that Dr. Roussy had emphasized clearly and had well illustrated that the emotional background, or the emotional dynamic factor, was the paramount situation in practically all of these cases. There have been many discussions on this subject. Dr. Jelliffe was a believer in the intrinsic effects of emotional situations, and had emphasized frequently that many disease pictures which have heretofore been called organic start at purely emotional levels, and ultimately, through long fixation and by long unconscious repression of affective material, arrive at organic goals. It was gratifying to hear that after a long period of time, eighteen months in one case and seven months in another, the hypertrophic changes in the skin, nails, sweat glands, and hair cleared up through rational psychotherapy. Dr. Jelliffe thought it remarkable that in so short a time such splendid results were obtained with this type of clientele. It is a matter of extreme regret, on the other hand, to recall how refractory so many similar conditions had proved in some of his own patients. And this stimulated him to attempt to arrive at some deductions concerning the mensuration of these emotional forces, about which Dr. Roussy mentioned certain experimental measurements of the amount of perspiration, and of oscillations of blood pressure conditions.

Dr. Francis X. Dercum, Philadelphia, said that it was noted by the English physicians that soldiers presenting functional nervous symptoms usually improved when sent to England, but relapsed if ordered back to the front, and at times, even before they got to the channel. In this connection the report of Surgeon-General Ireland, that out of 2,300 cases of functional neuroses—shell shock—2,000 recovered on the day of the armistice, is very significant. It would appear that when all prospect of reentering active service disappeared, the cases recovered. These cases had a parallel in civil life in the cases of hysteria following accidents, in which there were claims for damages. No improvement results until the question of damages has been disposed of, in one way or another. He understood from Dr. Roussy that the soldiers who were treated successfully were men who had not been in active service for some two years and who had practically no prospects of returning to the trenches.

Dr. William G. Spiller, Philadelphia, said that the pictures which Dr. Roussy had shown were exceedingly instructive. Especially interesting was the method of making the paralyzed hand move synchronously in the same kind of movement as that performed by the unaffected hand. A similar result might be obtained in organic conditions. This method of treatment may be employed after paralysis from a lesion of the central nervous system, or where there has been recovery from paralysis without return of function despite removal of the cause of the paralysis. Oppenheim called this condition akinesia amnestica. The nervous system seems to have forgotten how to make use of its function, if it may be so expressed. Dr. Spiller had at the time a case of peripheral facial palsy in which the benefit of this method of treatment was shown. The man had a severe organic facial palsy. He was made to draw up the nonparalyzed corner of his mouth, and each time he did so the galvanic current was passed on the paralyzed side of the face, and considerable power had been regained.

Dr. Daniel J. McCarthy, Philadelphia, believed a slightly different interpretation could be placed on these cases than that which Dr. Jelliffe gave. In a study of a number of cases of this type in 1914 and 1915 at the American Ambulance, and in 1918 and 1919 at the Neurological Institute at Vichy, a definite number of cases fell into a group which explained to a certain extent the bilateral symptoms. He assumed that in cases in which there was very intense injury, for example, there were physiologic reactions not only on the same side but on the opposite side as well, and in studying a series of intense injuries of the bones of the shoulder girdle it was found that particularly after these accidents there were trophic disturbances on both sides. In rare instances there was clear cut delimitation of function and sensation confined to the injured side. It is a vast problem, and it should be remembered that the question of a separation of what might be called functional overflow and actual organic disease must be dealt with. Dr. McCarthy believed that in this type of case there is a definite reaction with certain vasomotor and trophic disturbances, which, if the individual had sufficient purpose for remaining out of the service and if he was not properly examined in the beginning, would have become apparently organic. At least this condition obtained in Dr. McCarthy's service. There were approximately 1,000 cases at the Institute of Vichy, but very few of the intensity of Dr. Roussy's cases, because the patients realized that a clear cut elimination of the functional group was made, and therefore the symptoms cleared up. Those cases which one might interpret as the segmental group, with intensive injury of nerves, muscles, or bones and clear cut symptoms on the other side, did not prove persistent, except in a few instances. Dr. Roussy's presentation was extremely interesting but the interpretation is open to question. The primary condition is first organic, and secondly reflex on the vasomotors, affecting rarely the opposite side. What might be called hysteria, or defense reaction, kept the individual out of the service with persistent symptoms. Hysteria must here be used in the broadest sense of the term. Any man who has been through front line service would not be glad to return, and if he knew that an injury of the hand would be a decisive factor in postponing his return, he would try to prolong his symptoms until the end of the war. On the other hand, in contradistinction to such cases, there were those soldiers who, as soon as they found that they were dealing with diagnostic tests, realized that they would have a difficult time of it, and got well.

Dr. Gustave Roussy, in closing the discussion, said that in regard to the question brought up by Dr. Dercum, it was a very difficult matter to give absolute statistics or percentages, but approximately in from one tenth to one fourth of the cases, there was actual failure, either partial or complete, in obtaining therapeutic results. The remaining three fourths could again be subdivided; some of them were completely cured and returned to active service, but the greater number had already had disturbances for several months or years. These soldiers were not discharged entirely, but were placed in what was known as the auxiliary service and kept under military discipline. The next point emphasized was that the various neuroses which developed during the war were psychologically analogous to those observed in civil life, but in interpreting the ease with which the therapeutic result was attained, one had to remember the extraordinary prestige and influence of the military physician, who was not only a physician, but likewise a military officer. Furthermore, everything was so arranged that all cases could be grouped under one roof, so to speak, or in one fairly well compassed area subject to the general supervision of one medical and military head, so that

all privileges and disciplinary measures were absolutely regulated. The men were practically obliged to submit integrally to the various measures adopted for the relief of their symptoms. Unquestionably some of these cases were cured much more promptly after the cessation of hostilities, but an important fact was that even at that time a large number of patients failed to recover spontaneously. They had to be helped by some therapeutic measure. In regard to Dr. McCarthy's question as to whether the various motor disturbances were not primarily organic and secondarily functional, the opposite view had been assumed by him, by reason of the experiences which he had had, namely, that the motor disturbances were functional from the outset, inasmuch as if treated at once they were invariably cured completely and no secondary trophic or vasomotor disturbances appeared.

REPORT ON THE STUDIES OF DR. R. S. ELLIS TOUCHING THE STRUCTURAL CHANGES IN THE HUMAN CEREBELLUM—FROM BIRTH TO OLD AGE—TOGETHER WITH A QUANTITATIVE DETERMINATION OF THE PURKINJE CELLS IN NORMAL AND SUBNORMAL CEREBELLA. Dr. Henry H. Donaldson.

Between birth and 15 months the external layer of granule cells disappears and this event coincides with the appearance of the ability to walk. The relative weight of the human cerebellum characteristic for the adult is attained at about 15 months, and this relative weight is not significantly modified by stature, sex, age, race or grade of normal intelligence. After the thirtieth year there begins to appear a systematic loss in the number of Purkinje cells and this becomes more marked in old age. There is some evidence to show that this loss is usually greater in the right cerebellar hemisphere. In idiots and imbeciles this loss is very marked and in them some correlations have been made between the localities of the greater deficiencies and the type of motor disturbance.

DISCUSSION

Dr. Harold N. Moyer, Chicago, asked if there was any relation between right handed and left handed individuals in the results obtained in these studies.

Dr. Edward D. Fisher, New York, asked what the relative difference was between the cerebral cortex and the cerebellum in regard to the disappearance of cells as individuals grow older.

Dr. Donaldson, in closing the discussion, answered that he had no data to present on the cerebral cortex. As to Dr. Moyer's question, unfortunately he had not the records as to whether the individuals were right handed or left handed, but he assumed that right handed individuals predominated. He added that the volume of the full Purkinje cell was attained in about two years.

RARER CLINICAL TYPES OF EPIDEMIC ENCEPHALITIS. Dr. Bernard Sachs. This paper will appear in an early issue.

This paper is a presentation of some of the more unusual forms of epidemic encephalitis, with special reference to those presenting meningeal and psychic symptoms. A few other cases are also given to prove the justice of speaking of these epidemic cases as "central or basilar encephalitis."

FURTHER STUDIES ON THE ETIOLOGY OF ACUTE ENCEPHALITIS. Drs. Israel Strauss and Leo Loewe.

Cultural characteristics of the organism. Animal inoculations. Study of the nasal washings and spinal fluid and the use of these as a means of diagnosis.

THE TENDENCY OF EPIDEMIC ENCEPHALITIS TO CHRONICITY. Dr. Isadore Abrahamson. This paper appears in this issue.

CHOREO-ATHETOID AND CHOREOPSYCHOTIC SYNDROMES AS CLINICAL TYPES OR SEQUELAE OF EPIDEMIC ENCEPHALITIS. Dr. LaSalle Archambault. This paper will appear in an early issue.

Epidemic encephalitis is characterized by a protean symptomatology, the variations of which are dependent on the location of the predominant histologic lesion. In addition to the usual and familiar clinical picture of midbrain involvement, the disease may be manifested in a number of clinical types closely resembling such diseases as paralysis agitans, poliomyelitis, polio-encephalitis, polyneuritis, cerebellar sclerosis, hemiplegia and others. Aside from the disparity in clinical manifestations, there are variations in the course and evolution of the disease. The disease may be of acute or subacute onset, and its duration may be very variable, even to extending over several months. After the complete disappearance of a few abortive manifestations of typical encephalitis, certain choreiform twitchings and acute psychotic disturbances are not uncommonly seen as the salient manifestations, and choreo-athetoid or frankly athetoid syndromes may be observed as sequelae of the disease. The occurrence of a chronic or relapsing form of epidemic encephalitis may have an important bearing on the origin of some of the affections simulated by the clinical types of the disease and of such obscure conditions as multiple sclerosis, hypothalamic and dysglandular syndromes, and certain psychoses, including perhaps dementia praecox. The variations in the symptomatology are possibly due to individual variations in the relative susceptibility of different levels of the cerebrospinal axis; regional exhaustion from prolonged hyperactivity or previous disease or trauma is an important factor in the localization of the lesions produced by the infection of the central nervous system. To this same factor may be ascribed the frequent involvement of the mesencephalon in epidemic encephalitis with its resultant lethargy and oculomotor paralysis; probably never before have people slept so little and used their eyes so much as within the last five years.

DISCUSSION ON PAPERS OF DRS. SACHS, STRAUSS AND LOEWE, ABRAHAMSON AND ARCHAMBAULT

Dr. Daniel J. McCarthy, Philadelphia, in reference to the incidence of encephalitis in this epidemic as compared with the epidemic of 1889-1890, in contradistinction to Dr. Sachs' experience, recalled that in those days there was a very definite group of cases not described as sleeping sickness, or as encephalitis, which conformed very closely to the type seen at the present time. In those days, the cases came to attention two, three or four years after the original epidemic. There were milder epidemics during the following two or three years. As he remembered them, they were very often of a severe type with spinal symptoms.

With reference to diagnosis, in the Army in France there was an epidemic of influenza, but it was not as intense as the epidemic in this country. He remembered Dr. Cushing calling his attention to the ephemeral nature of the early ocular cerebral symptoms. The ocular palsies were very transient and lasted only one, two or three days, and then sometimes returned accompanied by definite somnolence and more fixed symptoms. In those cases the principal feature was the extremely slow development of the symptoms; a patient might go along for two, three or four weeks before the diagnosis was finally established and he reported sick for examination. He had a very slight attack of this condition himself and went through a period of somnolence with only one symptom of cortical irritation affecting the muscles of one hand. As one follows the pathology of this disease there is no necessity, from his standpoint, to make any remarkable divisions of the distribution of the lesions in epidemic encephalitis, since these distributions may be extensive and widespread, and in that way leave a general picture of the disease as one sees it. The diagnosis can be very simple without an arbitrary classification.

Dr. Adolf Meyer, Baltimore, asked for the opportunity of demonstrating a few lantern slides which would direct attention to one variety of the disturbance that has not specifically been named—pseudotumor. They dealt with a patient who showed symptoms suggesting encephalitis in the early process of the disease, and who, about three months afterward, began to show some head symptoms—choked disk, complete blindness, complete deafness, with a certain amount of staggering. The patient died during an operation for decompression, and showed, in reality, a diffuse meningitic process, with a peculiar type of cell infiltration. With all these apparent pressure symptoms and the tremendously diffuse meningitic and encephalitic process, the patient showed a perfectly astounding mental clearness and ease.

The brain showed practically no evidence of macroscopic disorder, except a little asymmetry and hemorrhagic discoloration of the uvula of the cerebellum and the choroid plexus and slight milkiness of the pia of the convexity of the cerebrum. Under microscopic examination there were found a peculiar infiltration of the meninges of the base, the cerebellum and the cerebrum, and a few encephalitic foci in the hind-brain and cerebrum, with a large cell type, reminding one of the cells Essick found under mild irritation of the pia arachnoid, only much larger, often pear-shaped and epithelioid, with total absence of the characteristic small lymphoid type of infiltration. The chief points in the case were the symptomatology of pseudotumor with choked disk, deafness, blindness, remarkable mental clearness of the patient and the diffuse meningitic infiltration with this peculiar cell type; hence the propriety of speaking of a meningo-encephalitis magnocellularis.

Dr. Harold N. Moyer, Chicago, said that the remark of Dr. Sachs regarding the ease with which a correct diagnosis may be made during an epidemic suggested that a wrong diagnosis may be made with equal facility. In a well marked case reported by Dr. Bassoe presenting a typical picture of this disorder, including the temperature changes, was found to be a case of tumor of the midbrain. A patient in Dr. Moyer's service at the Psychopathic Hospital presenting a typical clinical picture of the lethargic form was found after death to be suffering from a basal skull fracture.

Dr. Colin K. Russel, Montreal, said that one of the very interesting points in this epidemic has been the symptom of somnolence, chiefly because, in his experience at least, it was practically seen only in cases where the clinical evidence pointed to a lesion in the posterior part of the pons, giving rise to

ocular symptoms, particularly nystagmus. He had seen this symptom also in a young patient in whom the clinical evidence of laryngeal paralysis and paralysis of the muscles of the neck pointed to a lesion in the lower part of the medulla and upper cervical cord. Somnolence in this case was marked and lasted over a week. On the other hand, in a patient with an apparently uncomplicated bilateral facial paralysis, there was no history of somnolence. The interesting suggestion had been offered that this symptom might be due to involvement of the pituitary gland, but in one of his necropsies, in a case with marked somnolence no pathologic lesions of this organ were found. Dr. Russel's experience rather inclined him to agree with Farquhar Buzzard's suggestion that the somnolence might be due to a closing off of the aqueduct of Sylvius giving rise to disturbances in the circulation of the cerebrospinal fluid and an acute hydrocephalus.

Dr. William G. Spiller, Philadelphia, said that the short duration of the ocular palsies was characteristic. They come rapidly; in one patient the first symptom noticed was double vision in looking out of a window. For this reason the ocular palsies might be overlooked. This is also true of other cranial palsies. He had seen facial palsy lasting only a few days. Regarding Dr. Archambault's observations about the choreic movements, Dr. Spiller said there was a patient in the University Hospital, sent by Dr. George Wilson, who had had these choreic movements since the epidemic of 1918. Dr. Spiller had come to believe, although possibly he had not sufficient basis for this conclusion, that the choreic or myoclonic form of lethargic encephalitis was caused by a lesion in the region of the basal ganglions rather than by a lesion near the aqueduct of Sylvius. He referred to that in his paper before the Association the previous day. Dr. Spiller states he did not know of any necropsies that had been reported in the myoclonic type. He had a case in March of this year of a man who had had this myoclonic type for several weeks. He admitted him to the hospital, and not supposing there was any immediate danger, expected to see him the next morning, but before he arrived the patient had died. In the necropsy he found that the perivascular infiltration was slight in the region of the aqueduct of Sylvius, but that there was considerable infiltration throughout the basal part of the temporal lobe.

In regard to the somnolence, this symptom seems to be more striking in cases in which the region around the aqueduct of Sylvius is involved. It has been the view of some that the optic thalamus is the recipient of all afferent impulses, and when it is diseased the impulses are unable to reach the cortex and lethargy results.

About October of 1918 Dr. Spiller had a case in his service which was reported by his intern, Dr. Smith, and published in the Archives of Neurology and Psychiatry, and which seemed at first to be one of tumor. It was the case of a young boy who had choked disk, ocular palsies and a paralysis of the soft palate. It was very difficult to explain this combination by a diagnosis of tumor. It was not tumor and complete recovery occurred. The case was regarded as one of encephalitis simulating tumor, and Dr. Spiller referred to it later in the same journal under this diagnosis.

Dr. Israel Strauss, New York (in closing discussion), said for both Dr. Sachs and himself, that their experience at the hospital covered a total of something over a hundred cases, and he had seen in private practice at the lowest estimate about fifty additional cases. They have observed practically every form of this disease. The French have begun to describe a large number of syndromes, but all of these have been observed in this country.

They have also observed the transient palsies that Dr. McCarthy mentioned, and which they regard as abortive types. Regarding the cases following the

influenza epidemic of the nineties, Dr. Strauss said he could not speak from personal experience, but he would like to call Dr. McCarthy's attention to the fact that at that time a great number of cases of acute encephalitis were described and were supposed to be influenzal encephalitis. In their work they found one strain, which in the beginning produced the picture of hemorrhagic encephalitis, and as they transmitted that virus through animals its character changed so that it produced the histologic picture now regarded as typical of lethargic encephalitis.

Another interesting fact noted was that in one of the cases of an acute maniacal state, with myoclonus, the organism was obtained. Strangely enough, those patients sometimes, without any symptoms of palsy, were the ones that died very soon.

Dr. Strauss stated that he thought it hazardous to draw conclusions regarding the site of the region involved in symptomatology, from the extent of the lesion found in any part of the brain. They had performed a large number of necropsy examinations and had been struck with the fact that in some cases the extent of the lesion was remarkable, the entire basal ganglions being affected in toto, while in other cases, showing precisely the same symptomatology, just as severe in type, they were amazed to find such a small lesion. Dr. Sachs had referred to the fact that one can account for the severity of the disease by the severity of the lesion. For this reason he believed it would be hazardous for anyone to postulate on what is found in the brain any theory regarding the localization of any given symptoms.

Dr. LaSalle Archambault, Albany (in closing), said that from the beginning the papers published on this disease had given a rather confusing impression of the condition of the spinal fluid. Many series of cases have been published in which the fluid had been almost universally negative. Dr. Archambault said that had not been his experience at the Albany Hospital. Definite changes were found in the majority of cases. He was unable to state the exact percentage, but it was possibly 60 to 70 per cent. The findings were not pronounced, but definite. The cell count varied from 8 to 200 cells per cubic millimeter, and the globulin was positive, the reaction being reported as two or three plus. In some cases there was a peculiar reaction with the colloidal gold test; the discoloration occurring in the first tubes somewhat suggested the findings in syphilis or general paresis. The Wassermann reaction was negative in both the spinal fluid and the blood.

As Dr. Strauss had brought out, there were many cases diagnosed as encephalitis, and other cases which remained undiagnosed. Eventually we shall have more accurate means of diagnosis, but in the meantime we are not all fortunate enough to have Dr. Strauss with us, and in the absence of such positive scientific data as he would be able to furnish, other available means must answer. Dr. Archambault believed and would like to go on record as stating that, while not absolutely essential or necessary, changes such as he had described in the spinal fluid were of extreme value in helping to make a proper diagnosis in epidemic encephalitis.

CEREBRAL SURGERY. Dr. Harvey Cushing.

In two interesting and suggestive papers Weed and McKibben[1] demonstrated the significant physiologic fact that it is possible to reduce the cere-

1. Experimental Alterations of Brain Bulk, Am. J. Physiol. **48**:531, 1919; Pressure Changes in the Cerebrospinal Fluid Following Intravenous Injection of Solutions of Various Concentrations, ibid., p. 512.

brospinal fluid pressure and diminish the bulk of the brain by injecting a hypertonic solution into the blood stream. Conversely they found that hypotonic solutions had the opposite effect: a rise of cerebrospinal fluid pressure and an increase of brain bulk. In the course of our studies their work has been repeated and their general conclusions have been confirmed. The clinical bearing of these facts is obvious. They concern particularly the states commonly referred to as "pressure symptoms." By similar methods it has been found possible to secure the same results in patients with increased degrees of intracranial tension. It was felt that the undesirable effects on pulse, respiration and blood pressure of such intravenous injections might contraindicate their use. For this reason the effects of gastro-intestinal doses of hypertonic solutions were studied.

In a large series of animal experiments it has been found that practically the same effects may be obtained by gastro-intestinal administration. By this method the intracranial changes are not attended by disturbances of pulse, respiration or blood pressure, and the possible alterations of the cellular elements of the blood are avoided. From 20 to 30 c.c. of a saturated sodium chlorid solution introduced into the duodenum or rectum of an average-sized cat produced a maximal fall of cerebrospinal fluid pressure precisely comparable to that which occurred when the solution was given intravenously. Following such doses the average fall of pressure in a large series of experiments was 258 mm. normal saline. The changes were roughly proportionate to the concentration of the salt and the size of the dose. Two per cent. sodium chlorid solution in large doses or a saturated solution in doses as small as 5 c.c. gave corresponding though less marked effects. Nonabsorbable salts gave similar reductions though slower in occurrence and less in extent. Dextrose solution caused results still less striking though identical in nature. The converse results seen after the ingestion of hypotonic solutions (water) were not great in extent nor well sustained. A previous dose of a hypertonic solution rendered more extensive the rise of pressure in these cases. The changes observed were independent of blood pressure and were not attended by significant alterations of pulse or respiration.

These effects on cerebrospinal fluid pressure and brain volume were investigated in human subjects, an excellent opportunity for this being afforded by patients with brain tumor and cerebral hernias subsequent to decompression operations. In these patients there was a lowering of tension when hypertonic saline solutions were given by mouth. Occasionally very striking results could be obtained, in which cases the tense convex protrusion became a soft concave area over the decompression site.

PUNCTURE OF THE CISTERNA MAGNA. Dr. James B. Ayer. This paper will appear in an early issue.

DISCUSSION OF PAPERS BY DRS. CUSHING AND AYER

Dr. Ernest Sachs, St. Louis, said that he had come to realize in the last few years that the handling of the cerebrospinal fluid was one of the most important factors in dealing with intracranial conditions, particularly tumors. Consequently, he had been greatly interested in the work of Weed and McKibben. Dr. Cushing pointed out in his paper that he had shown experimentally that saturated salt solution injected intravenously would reduce the volume of the brain. Dr. Sachs had tried this on several cases, just as Dr. Cushing had done. The result in the reduction of the hernia was so striking that one of

his assistants, Dr. Belcher, urged him to try it in the course of an operation. Since he had only Weed's experimental evidence to go on, he hesitated to use it except in a case in which it was impossible to get cerebrospinal fluid by ventricle puncture, as happens not infrequently in supratentorial tumors, because these tumors often compress the ventricle. In these cases opening the dura without reducing intracranial pressure is likely to meet with disaster. He recently tried this, therefore, in the course of an operation and it gave satisfactory results, and although it was only a single observation, it seemed worth recording as the method promises to be of great value.

This particular case was one of temporal lobe tumor in which both ventricles were aspirated during operation to reduce pressure, prior to opening the dura, and in which both the right and left ventricle were completely compressed and only a drop or two of fluid was obtained. There was then introduced, intravenously, 30 to 35 c.c. of a 35 per cent. saturated solution of salt and within five to six minutes the brain had contracted to such an extent that it was possible to open the dura with the brain practically under normal tension. Studies were made in this case for a number of days afterward to determine whether there was any harmful effect from the salt solution and also whether the fragility of the red blood cells might have become affected, a point that Dr. Marshall, our pharmacologist, was much interested in. The studies made in this one case showed that the fragility of the red blood corpuscles was not impaired in any way, and as far as they were able to observe, there was no deleterious effect on the patient.

It seemed to Dr. Sachs that this procedure during the course of intracranial operations was one that must be undertaken. He undertook it with much hesitation, but felt that the experimental work of Weed and McKibben justified it in an extreme case.

Regarding the work of Dr. Ayer, he felt as most surgeons did who did not like to undertake procedures in the dark. He would fear greatly to undertake such a puncture of the brain cisterna. He noticed in his drawing that Dr. Ayer did not take into consideration two points which, it seemed to him, must be remembered: First, the medulla is suspended in fluid and, therefore, during life it may be much nearer the posterior portion of the canal than it is in the cadaver. The cord and medulla move markedly during respiration. During inspiration the cord is forced back, as can be seen in the course of any spinal operation. And, second, not infrequently there is a vein of considerable size that runs from the torcular down the median line in the dura, and Dr. Sachs thought there would be great danger in some cases of puncturing that vein since, according to Dr. Ayer's drawing, the needle is passed so close to the posterior edge of the foramen magnum that where this vein ends in the circular vein around the foramen it may readily be injured. No one cares to have blood in the region of the medulla; surgeons try to avoid having even a drop there.

Dr. Harvey Cushing, Boston, thought there could be no doubt but that Dr. Ayer had realized the risks of this procedure as much as any one.

In regard to what Dr. Sachs had mentioned, Dr. Cushing said that this particular median vein stopped at the foramen, so that there was no risk on that score. Nevertheless, the procedure is a dangerous one. However, he cautioned that Dr. Ayer was merely reporting some preliminary studies, and even lumbar puncture under certain circumstances is dangerous. He was quite sure that the procedure which Dr. Ayer had in mind may, with due precautions, become useful. He thought the same thing was true of the saline procedure, of which he had spoken.

Dr. Cushing said it must be realized that we are in the early stages of these investigations, and it was long after Quincke's time that lumbar puncture became routine. He was sure there is a great deal in all of these new procedures that may become valuable, but he believed that such a measure as Dr. Ayer advocated should not be promptly adopted by all physicians, as accidents would follow. The measure should be left in the hands of those who were particularly interested in it, to develop the technic until we could take it over from them and rely on it. He thought the same thing was true of injecting air into the ventricle for diagnostic purposes. The time had not yet come when any one of these three novel procedures could be advocated for general use.

Dr. Foster Kennedy, New York City, said that Dr. Ayer's description of the first step in his technic and his directions for finding the first cervical vertebra rather reminded one of the opening sentence of Mrs. Beaton's recipe for making Welsh rarebit—"First catch your hare."

The procedure that Dr. Ayer had touched on was one that ought to be viewed somewhat askance, and as Dr. Sachs wisely pointed out, its technic ought to deter us from undertaking it lightly. Surgery in the dark, as Dr. Sachs remarked, is, on the whole, rather a precarious procedure, and when in the neighborhood of the medulla, since the medulla is not a fixed point, it seemed to Dr. Kennedy to be doubtful. If it is a matter of removing cerebrospinal fluid from the great cistern of the brain, and one has the choice of tapping the cistern, or giving saline by mouth, it seemed better for the patient to take the salt!

As for the suggestion that this method is a proper one with which to treat cerebrospinal syphilis, he said that this has not yet been demonstrated.

Dr. Adolf Meyer, Baltimore, called attention to a way of getting a good view of this region with a procedure that suggested itself to him because he desired to obtain more perfect specimens at necropsy. The attempt to obtain a well preserved position and normal topographical relations of the brain led Dr. C. B. Dunlap to work out a technic of formaldehyd injection before removal of the brain at necropsy. This made of the brain a relatively inelastic mass; hence the necessity of adding to the usual circular section of the skull the removal of a wedge of the occipital bone, exposing the foramen magnum and under the dura the pia arachnoid of that region.

Having thus had the frequent opportunity of seeing that region in formaldehyd injected material under the best possible exposure, so that we were not in the dark, Dr. Meyer was decidedly impressed with the fact that we were dealing with a space much wider and more easily reached than he would have dreamed. The tapping of the posterior tent of the pia arachnoid was, really, as Dr. Ayer described, much safer and easier than any one who had not performed that necropsy technic would consider likely. He thought it wise, however, to consider the possibility of cerebellar herniation.

Dr. Bronislaw Onuf, Rutherford, N. J., wished to state that he had applied Dr. Ayer's method in about fifty cases with entire safety, meaning that as it was used postmortem, the danger of causing death was excluded.

He had previously referred to a method which he wished to speak of again because the subject had some bearing on Dr. Cushing's paper. Some fifteen years ago he described a method of preserving the brain and spinal cord after death without performing necropsy. He was, at the time, pathologist to Craig Colony, and in many cases he had to wait twenty-four to fifty hours after death for permission to perform the necropsy examination. In most of these cases

the brain and cord would have been absolutely annihilated for microscopic study if something had not been done to preserve it before the necropsy was made. A method was devised which was described in the *Medical Record* (July 9, 1904) of injecting a solution of formaldehyd, 12 per cent., through two routes—one by lumbar puncture and the other through the cisterna magna. The injection was made under pressure, and he was surprised at the large amount of fluid that could be injected in this manner. Usuaully, only about 400 c.c. could be so injected, but he had had cases in which as much as 1,500 c.c. went in. The fluid oozed out along the nerve roots, hardening the parts adjacent to the spine, lungs, liver, kidneys, intestines, etc. That the fixation was a success was shown by applying to the basal ganglions such delicate tests as Weigert's and Mallory's neuroglia stains which succeed only in specimens hardened almost immediately after death, and which in the specimens made from the basal ganglions of brains hardened by the prenecropsy method mentioned, gave perfect results.

The point of interest with regard to Dr. Cushing's investigations was the oozing out of the fluid along the nerve roots, showing some of the pathways along which the cerebrospinal fluid may wander.

Dr. Israel Strauss, New York, thought we ought not to allow this piece of work of Dr. Ayer to pass without some commendation instead of criticism. Dr. Ayer had not only experimented on animals—eleven hundred—but on the cadaver, and his results were based on experimental knowledge.

When it came to "working in the dark" he believed that some of the procedures, like that advocated by Dr. Ayer, were safer than some others in which surgery worked in the light. He believed he would take chances with this procedure rather than allow general surgeons to attempt to aspirate the ventricles, because he had seen experts fail even in this.

As Dr. Cushing had stated, there were dangers in lumbar puncture and there were dangers in every other procedure. He had seen a patient die from an ordinary injection of one-hundredth of a grain of hyoscin, and yet this drug was in general use.

There was only one warning that ought to be given regarding Dr. Ayer's work and that was that before any of us, or the surgeons who "worked in the light," attempted this procedure, they should first try it on the cadaver. This is the particular point to which Dr. Ayer should call attention when publishing this paper, namely, asking physicians to first try it on the cadaver, and not to discredit a procedure as harmful which was of great value when properly used.

Dr. James B. Ayer (in closing) said he did not expect the acceptance of this procedure without remonstrance, and he was more than pleased that so many thought there was anything of value in it. He, too, realized the potential dangers of cisterna puncture, but believed that if used carefully there would be no harmful results.

There was another group of cases in which cistern puncture should be of use. In septic meningitis, for which there was at present no adequate treatment, irrigation from cistern to lumbar sac, or ventricle to cistern or cerebral subarachnoid space to cistern, offered a method which may well be tried. As to the serum treatment of cerebral syphilis by this route, he was not sanguine, but if there was value in serum treatment it should unquestionably be of greater value if given by this rather than by the lumbar route.

In finding the point for insertion of the needle, the landmark was not the spine of the first, but of the second cervical vertebra, which is readily palpated on most subjects. Points in technic were necessarily made brief because of the time, and because this had already been published.

The conception of the medulla being freely movable did not seem at all likely. In a cadaver in which a needle was left in situ, the brain was removed until the medulla and needle were visible; the head was then thrown into different positions; in no attitude could the needle be made to impinge on the medulla.

CHICAGO NEUROLOGICAL SOCIETY

Regular Monthly Meeting, May 29, 1920

LEWIS J. POLLOCK, M.D., *President, in the Chair*

DEMONSTRATION OF A BRAIN WITH DEEP BRUISE AND A BRAIN WITH PORENCEPHALUS. Presented by DR. E. R. LECOUNT (by invitation).

Dr. LeCount presented a brain with a deep bruise and hemorrhage which closely simulated the lesions of apoplexy, and exhibited charts showing a fracture of the right side of the skull. With these injuries there had been marked compression from an extradural hemorrhage. He stated that he had seen few deep bruises of the brain and that much bruising of the brain of any sort with extradural hemorrhage was uncommon.

The patient was a man of 25 years, and death had occurred eleven days after the injury. Dr. LeCount referred to the possibility of the deep hemorrhage being secondary, a so-called late traumatic apoplexy.

The brain with porencephalus was from a large negro, 6 feet tall and weighing 160 pounds, removed from a car on the Illinois Central during the winter with frostbitten feet and legs and taken to the Hospital of the House of Correction, where he died within a few hours, without any symptoms referable to the brain. Dr. LeCount thought that the malformation of the brain caused death because of the marked edema of its substance; moreover, postmortem examination failed to disclose disease elsewhere adequate to explain death. The brain was flattened and the frontal lobe, on which the defect was located, was adherent to the dura on the right side. When this adherent dura was lifted up a little a clear fluid, now opaque from liquor formaldehydi, could be seen which extended into the right ventricle. It is not an uncommon maldevelopment, 200 or more being reported, but so far as he knew the brains had been studied only as to the maldevelopment. In his opinion, the brain should be sectioned and studied, not for an explanation of the defect, but for its usefulness in connection with special problems of nerve pathways through the brain, still unsolved.

PARTIAL OCCLUSION OF THE RIGHT POSTERIOR INFERIOR CEREBELLAR ARTERY. DR. GEORGE W. HALL.

A man, aged 34, married, father of one healthy child, gave no history of illness of importance; he had never had syphilis; his use of liquor had always been moderate; he smoked about six cigars a day. No history of miscarriages.

On April 19, 1920, the patient noticed a "cool" taste in the right side of the mouth. On April 20, while attempting to get on a street car he fell to the right and lost out of his right hand the money which he had ready to pay his fare. On April 23, a similar cool feeling gradually spread over the right side of his face, more noticeable on the lower lip. On April 24 he had a slight attack of vertigo when changing his posture from horizontal to vertical. On April 26 he noticed some difficulty in controlling movements of the right arm. The right leg was normal. On the same day he had some difficulty in articulating and also had slight blurring of vision which cleared up within a day or two.

Examination on April 27 showed blood pressure to be systolic 125; the urine, heart and lungs were normal and there was no evidence of gastro-intestinal disturbance.

There was distinct sensory disturbance over the right half of the tongue and right side of the face and slight narrowing of the right pupil and palpebral fissure. Both pupils responded to light and accommodation. There was no diplopia, but when the patient looked straight ahead horizontal nystagmus was present and became exaggerated on looking to the right and left. There was no paresis of the facial muscles, no deviation of the jaw on opening the mouth and no disturbance of hearing; the pulse was normal. The tongue showed no deviation. Except as noted above, sensation was normal. The abdominal reflexes were normal; the knee jerks were lively, there was no Babinski reflex and no ankle clonus. When the patient attempted to rise from a sitting position there was a tendency to fall to the right; he stated that the room seemed to go to the left, but his body did not feel as though it were moving. The tendency to go to the right was exaggerated when walking with eyes closed. There was no ataxia of the hands.

On April 28, while attempting to lift a glass of water to his mouth with the right hand, the glass slipped from his grasp and fell to the floor.

The blood Wassermann was negative.

The symptoms above described remained about the same until May 3, when they gradually disappeared. On May 12 there were no numbness and dizziness. Examination on May 20 revealed nothing abnormal.

Report of Otological Findings (by Dr. J. Gordon Wilson).—Lying in bed. No vertigo. Nystagmus: horizontal, slow to right; range small; diminished if he looks to right; increased if he looks to left. Pointing: right hand, touches up down, and down up; left hand: touches up down, and down up. Rising in bed: Vertigo—feeling of instability, not rotation. Sitting up: Nystagmus and pointing as before. Hearing: Normal; no tinnitus.

Conclusions: (1) Labyrinthine nystagmus, (2) vertigo on displacement of head, and (3) no acoustic involvement.

Caloric.—April 30, 1920. Right Ear (7 ounces of water at 58 F.). Head forward 30 degrees, vertical canal: Nystagmus marked, slow to right, rotatory. Pointing: right touches; left touches. Vertigo, slight. Head back 60 degrees, horizontal canal: Nystagmus marked, slow to right, horizontal. Pointing: right touches; left touches. Vertigo slight.

May 3: Right ear (water, 20 ounces at 57 F.). Superior canal, nystagmus, slow to right; rotatory 60 seconds. Horizontal canal, nystagmus, slow to right, horizontal 90 seconds. Pointing: touches with right and left after both superior and horizontal stimulation.

April 30. Left Ear (5 ounces water at 58 F.). Head forward 30 degrees: Nystagmus, slow to left, rotatory. Pointing: right to left, 3 inches; left to left,

3 inches. Vertigo slight. Head back 60 degrees: Nystagmus, slow to left, horizontal. Pointing: left to left, 4 inches; right to left, 3 inches. Nystagmus persists longer in horizontal plane.

May 3. Left ear (water, 8 ounces at 58 F.). Superior canal, nystagmus slow to left rotatory, 75 seconds. Horizontal canal, nystagmus slow to left horizontal, 130 seconds. Pointing error to left marked. No vertigo.

Summary: (1) Nystagmus present after stimulation of right and left labyrinth—normal in character but more easily produced with longer duration in left side. (2) Vertigo—very little, if any. (3) No pointing error in right—neither from perpendicular nor horizontal canals. Marked pointing error in left—both from perpendicular and horizontal canals.

Rotation.—Ten times in 20 seconds. Horizontal Canals—to Right:—Nystagmus—slow to right—33 seconds. Pointing—touches with right and left. Vertigo—very slight, if any. Twice repeated after interval. Nystagmus—slow to right—45 seconds; to right—30 seconds. Pointing—right hand—marked pointing error in both occasions. Pointing—left hand—marked pointing error in both occasions. Vertigo—slight on first repeating, none on second.

Vertical—to Right. Nystagmus—slow to right—48 seconds; violent fall to left. Repeated. Nystagmus slow to right, 40 seconds. Pointing—right 5 inches to right; left 7 inches to right. Violent fall to left. Vertigo—none.

Horizontal Canals—to Left.—Nystagmus—slow to left—19 seconds. Pointing—right 2¼ inches to left; left 2¾ inches to left. No vertigo. Twice repeated after interval. Horizontal—to Left.—Nystagmus—slow to left, 15 seconds; to left, 20 seconds. Pointing—right about 3 inches to left; pointing touches. Pointing—left about 2 inches to left. Vertigo—none; vertigo—slight.

Vertical—to Left.—Nystagmus—slow to left, 36 seconds. Pointing—right and left—touches. Violent fall to right, a propulsion rather than a fall. No vertigo. Repeated. Nystagmus—about 5 seconds; slow to left. Pointing—right and left touches. Violent fall to right—a propulsion to right. Vertigo—slight.

Summary.—1. Horizontal canals: (a) Nystagmus markedly diminished on stimulating right labyrinth compared with left. (b) Vertigo little on either side. (c) Pointing: very irregular; absent once on each side; when present more marked on stimulating left labyrinth and here distinctly increased on repeated pointing; not so after stimulation of right labyrinth.

2. Rotation.—Vertical canals: (a) Stimulation of left labyrinth gave normal nystagmus; marked pointing error; slight vertigo. (b) Stimulation of right labyrinth gave diminished nystagmus; no pointing error; slight vertigo. (c) On stopping, violent propulsion of upper part of body in direction opposite to previous rotation.

HISTOPATHOLOGIC FINDINGS IN PSEUDOTUMOR CEREBRI. Dr. Percival Bailey (by invitation). This paper appears in full in this issue.

DISCUSSION

Dr. G. B. Hassin said that Dr. Bailey was able to show only a few specimens illustrating the lesions in his case. Under the oil immersion a still greater wealth of changes can be made out. These changes are decidedly in the nature of acute meningo-encephalitis which, however, can be found in any other type of acute or chronic inflammation of the brain (general paresis, lethargic encephalitis, trypanosomiasis). The difference is but in the localization of the changes. To determine the proper localization; a number of sections from

every part of the brain must be studied. In this case the temporoparietal lobe was found to be more involved than any other portion of the brain and it is, of course, impossible to tell whether the unequal involvement of the cortex is specific for such cases. Whatever the localization, Dr. Bailey's histopathologic and clinical studies definitely show that a meningo-encephalitis may give a clinical picture of brain tumor.

Dr. E. R. LeCount said that some years ago Pick of Prague introduced the term pseudocirrhosis of the liver, but there is no such disease of the liver and the term caused great confusion. Dr. LeCount questioned the advisability of introducing such a term as pseudotumor and thought it would be better to avoid confusion, to call it meningo-encephalitis with symptoms of tumor.

The island of cartilage in the dura interested him and he wished to know how large it was and whether there were many such islands. The dura is not supposed to make cartilage. He was also interested in the accumulation of cells about the blood vessels. He had not studied them but hoped some time to know the details of the staining according to the Schridde method of the cytoplasm of the cells that were said not to be lymphocytes. He had not been quite able to convince himself that accumulations of cells so commonly attributed to the migration of glia cells and their gathering in certain places, especially about blood vessels, were not in reality due to the passage of cells from the blood stream and collection subsequently at such places.

Dr. Peter Bassoe asked what Dr. Hassin and Dr. Bailey thought of the relationship of this sort of cephalitis to so-called lethargic encephalitis. Dr. Bassoe thought there was more hemorrhage than in lethargic encephalitis. In the latter the hemorrhages usually are recent, probably agonal but not with tissue reaction as in these specimens. In the present case there was an entirely different distribution of the lesions: little involvement of the brain stem and basal ganglions but considerable involvement of the parietal lobes. In his opinion Dr. Bailey's case corresponded more to what was previously described as influenzal encephalitis. He had not read the detailed description of so-called influenzal encephalitis, but he knew that Dr. Hassin had and wished to know whether this case was somewhat similar. He thought the case could not be included in the group of lethargic encephalitis; if so, it was the first case that had come to Dr. Bassoe's attention in which choking of the disk was present.

Dr. Julius Grinker stated that he had reported a case of encephalitis with choked disk in which the patient made an almost complete recovery. In the literature others had found choked disk.

Dr. Percival Bailey said he had tried only to establish the fact that this case, which clinically simulated a tumor of the brain, was an encephalitis. He was heartily in agreement with Dr. LeCount that the term pseudotumor cerebri had no justification. With regard to the islands of cartilage in the dura, he had tried to maintain throughout a purely objective description. They resembled more nearly cartilage than any other tissue. He left it to Dr. Hassin to answer the more technical pathologic questions but was aware that Tschistowitsch, working in Ziegler's laboratory, had demonstrated as early as 1898 active division of the glia cells in the repair of aseptic wounds of the brain.

Dr. G. B. Hassin said that Dr. Bailey, in his case, made a special study of the midbrain and substantia nigra, the regions that are always affected in lethargic encephalitis. In his case they were found practically normal. The hemorrhages in his case were small, microscopic and rare, and always accompanied by marked reactive phenomena. Such a type of hemorrhagic phe-

nomena does not occur in lethargic encephalitis. On the other hand, in cases described as influenzal encephalitis, hemorrhages are the prominent feature without reactive phenomena and other changes, like perivascular infiltrations, parenchymatous lesions, etc. Such cases, commonly known as hemorrhagic encephalitis, run a rapid course while in Bailey's case the duration was three months. An attempt was made to explain the hemorrhages in the case under discussion on the basis of the operation, but this was performed three months before the patient's death and the hemorrhages were not confined to the place of operation, but to remote regions; like the optic thalamus and pons. In short, the hemorrhages like the rest of the pathologic findings were not traumatic, but of some infectious inflammatory origin. It somewhat differs from other types of encephalitis (lethargic, general paresis) by the localization of the pathologic changes and the presence of preagonal hemorrhages. It remains to be seen whether other cases of so-called pseudotumor give findings similar to those in Bailey's case. The numerous small nuclei scattered in the brain tissue are glia cells, not lymphocytes. Dr. LeCount doubts whether they can, with absolute certainty, be differentiated from lymphocytes that emigrated from the blood vessels. Without proper staining methods, he thinks, a differentiation cannot be made. The differentiation between these two types of cells, lymphocytes and glia nuclei, is not made from their staining qualities, which are practically alike, but on the basis of their embryologic properties. The lymphocytes are mesodermal elements, the glia cells ectodermal. These elements do not mix with each other, but occupy territories strictly assigned to them. Thus, the lymphocytes are confined to the vascular system and the perivascular spaces, while the glia cells are confined to the brain tissue outside the blood vessels. This teaching, promulgated by Nissl and his school, proved to be correct and is universally accepted by neuropathologists.

Dr. Ralph C. Hamill said that in a specimen showing diffuse melanosis of the cord and basal portion of the brain, large cells considered to be glia cells because of the presence of glia fibers were seen to contain many fine granules. These granules, because of their staining reaction and because of other evidences of hemorrhages, were believed to be of hematogenous origin.

Dr. E. R. LeCount said the granules he had in mind were those of the cell bodies or cytoplasm. Lymphocytes had been studied according to their granulae reaction to stains and classified rather extensively by those working in general pathology. He did not know whether this had been done in connection with the brain and until it has, one should not accept a dictum as to their origin—and possibly not then.

THE PRESENT STATUS OF THE ABDERHALDEN REACTION.

Dr. Clarence A. Neymann.

Perhaps no serologic reaction, the Wassermann test of syphilis excepted, has caused such wide discussion as the Abderhalden reaction. Authors closely associated with Abderhalden reported specific results with the test. Other investigators, among them some of the best trained, reported nonspecific reactions in pregnancy and other conditions. Fauser was the first to apply the test in psychiatry and reported positive reactions for testis, ovary and other glands of internal secretion in dementia praecox. Van Slyke separated the method from the questionable factor of the permeability of the dialization tubes. A dialization tube, even though just tested, might become worthless during a test and permit the passage of protein, thus nullifying the value of

the Ninhydrin reaction. The quantitative aminonitrogen method for blood serum—which gives the amount of the aminonitrogen present in the serum, before and after incubation of such a serum with the protein against which protective ferments are supposed to exist—does not confirm the original premises of Abderhalden. Thus, notwithstanding the later protest of Abderhalden himself, we cannot consider his theory or reaction as established.

Notwithstanding these facts, the Abderhalden test was employed for testing about one hundred different serums. The work was done by Dr. Frances L. Dunham and myself in the Phipps Clinic, Johns Hopkins Hospital. We have not heretofore published the results as they seemed to approximate those obtained by many other investigators. The dialization method was used with the technic recommended by Abderhalden in his "Abwherferment," fourth edition. The serums of ninety-seven patients were examined. Of these, forty-four were classed by us as of the schizophrenic type, thirty-nine suffered from various mental disorders, eight were normal pregnancies and six normal persons, male and female. Our material was quite different from that encountered in state hospitals. Most of the patients were in the earliest stages of their psychoses.

As is well known, all shades of blue can be obtained with the ninhydrin reaction. For our purposes, which were essentially clinical, the only thing that concerned us was whether a reaction was positive or negative. Whenever the dializate of the control tube, as happens with some serums, was slightly colored, and the dializate of the tube incubated with substrate was definitely blue, the reaction was considered positive.

Thirty-two of the forty-four serums of the patients of the schizophrenic reaction type were positive. Twelve were negative to sex glands. This corresponds roughly to 73 and 27 per cent. There was, however, a certain "overlapping" of the reaction; that is to say, of the twenty-five positive cases also tested with placenta as a control, five gave faulty positive reactions with this substrate. There was, therefore, a definite tendency for the serums of patients of the schizophrenic reaction type to give a positive ninhydrin reaction with sex glands. The results obtained with the serums of thirty-nine persons not of the schizophrenic reaction type, thirty of which gave negative, nine positive reactions with sex glands, corresponds to about 77 and 23 per cent., respectively. This is about the inverse ratio found in the first group, and would seem to show a definite and undeniable difference in the biochemical make-up of the serum of most schizophrenic when compared with the serum of other mental patients. There seems to be no doubt that the serums of schizophrenic patients are less stable when incubated and dialized with various organs and glands, than the serums of other patients. This instability is more easily brought about by sex gland tissues than other organic tissues. We believe that there must be some factor as yet unknown on account of which the serum of such patients is especially prone to dialize.

Recently Dr. Retinger has striven to overcome the many technical difficulties by using celloiden sacks for dialization and by allowing the serum to react with several sets of substrata. In his 1918 publication of twenty-five cases, four of which were controlled by necropsy examination and two by operation, we can see only those points of interest which we have seen throughout the development of the test—striking results balanced by peculiarities which at times were just as striking.

The most recent serums sent Dr. Retinger for examination have been interesting. We have only sent serums of such cases as could be absolutely concontrolled. One negative reaction was reported as a large parietal tumor through necropsy by Dr. LeCount. The other serum from a man with manifest endocrine disturbances, cryptorchism, or possibly Froelich's syndrome, was reported as giving a reaction with pineal gland and pancreas. All other glands of internal secretion gave negative reactions. In our opinion, many of these glands were affected. Thus, even with excellent technic and extreme care, unaccountable results can be obtained. We must, therefore, conclude that the Abderhalden reaction is interesting, may at times point in the right direction, but is thus far valueless for diagnostic purposes in psychiatry.

DISCUSSION

DR. J. M. RETINGER: The poor results obtained by most of the later investigators of the Abderhalden test are due to faults of technic and lack of experience. If we speak only about its application to the diagnosis of pregnancy, we find the following sources of error:

1. Unreliable Dializers: The judging of the original parchment sacks is extremely difficult.

2. Toluol: Toluol used as antiseptic contains many acid impurities which interfere with the digestion and the ninhydrin reaction.

3. The Substrate: Not all placentae prepared in exactly the same way are equally sensitive; some are indigestible and some are oversensitive, both probably due to minute pathologic changes.

4. Interpretation of Results: Placenta is to a large extent vascular tissue. If the patient suffers from any disease of the blood vessels we would naturally obtain a positive reaction, which cannot be interpreted as digestion of the placental specific protein.

5. Some serums have enough amino-acids in solution to give blue color with ninhydrin, a fact that is rarely taken into consideration.

The presence of specific ferments and their specificity was proved by Abderhalden by these tests: Ninhydrin reaction, polariscope, refractometer, the Van Slyke amino-acid N method, and colorimetric method, using stained proteins as substrata.

The presence of specific ferments and their specificity was proved by Hirsch by the interferometer, by Kantorowitch by the biuret test and by Kottermann by a colorimetric estimation of iron split off from ferrated substrata.

In my own experiments with the ninhydrin test I have obtained perfect specificity. These ferments appear to be even more specific than Abderhalden himself asserts. The specificity is so great that histologically identical parts of the brain could be differentiated sharply, provided their function was different. This observation was substantiated in a series of over one hundred cases of gross brain lesions when the lesion could be localized properly with this technic. Naturally this technic cannot be considered as based on a wrong theory, accidental results or a constantly repeated error, because only four cases of the series were localized wrongly, and because these results were obtained after almost six years of experience in this work.

DR. NEYMANN (closing) said, regarding the question of substrata that he, while working in Heidelberg, took up this question of overlapping and found

reactions with carcinomatous substrata and pregnant serums. After preparing tissues he sent these to Abderhalden and had them approved by him and used these tissues with the precautions recommended by Dr. Retinger. The same overlapping was seen. The tissues used in the test with dementia praecox serums were prepared with the greatest care. In order to make sure that they did not consist solely of connective tissue they were stained and examined under the microscope. He thought Van Slyke had done this much better than he, and even then the theory did not hold good.

Book Review

THE PROBLEM OF THE NERVOUS CHILD. By Elida Evans, Introduction by C. G. Jung; Dodd, Mead & Co., 1920, pp. 299.

It should be fairly stated at the outset in a professional review of this book that it disclaims being a presentation for professional people and that it deals very little with nervous children in the medical sense. The gist of the situation seems to be that Mrs. Evans, through her experience "in the difficult and toilsome treatment and education of nervous children," has developed a fund of practical wisdom that has led her to write this book "to aid those parents who in the training or education of their children have arrived at the point where the methods already used have proved inadequate."

There are various parts of the book that one heartily wishes might be read and reread by parents—the parents that one comes to know when studying difficult children—but it seems much less certain that the book as a whole can be considered a desirable dose to prescribe for any given parents. There seems to be danger in the many indiscriminating doubts and fears that the reading of the book, if it is taken seriously, will engender in the conscientious concerning their own behavior as it affects their children. Pitfalls for the young mind appear on every hand and how to cultivate a terrain free from serious danger during the developmental period seems altogether an uncertain task. Indeed, it seems difficult to explain how so many children get through without obvious injury when all the paths to disaster that Mrs. Evans enumerates are considered. These poor, foolish, silly, selfish, despotic, domineering, busybody parents with their lack of reasoning power and their "past of ignorance," as Mrs. Evans depicts them, undoubtedly trammel the growth of their children's lives, and psychanalysis of the parent would often be the best prerequisite of treatment of the child. The main point set forth is that the behavior and attitude of parents hamper the finest growth of children rather than that out-and-out nervous disorders are caused thereby.

It is going quite a long distance further to intimate that dementia praecox may be caused by the untruthfulness of a mother or that the fear of punishment causes epilepsy. As a matter of fact, however, many of the most common nervous states of children are not considered at all in this work, and many of the cases cited seem to be merely those of difficulty in social reactions or adjustments just as we should expect, with these backgrounds of unwise parental conduct that she cites.

The reader is offered occasional easy sermonizing on various subjects—bachelors in clubs, conditions in Russia, labor troubles—that seem hardly indicated by the title. In many places the interpretations are clearly of the cocksure and seemingly far-fetched sort that users of symbolism and dream material indulge in.

However, nothing quite so sticks in the reviewer's crop as the social or economic implications of this work. Difficult children—and if possible their parents—are to be studied and treated by the regular psychanalytic method. Of course we read, then, of the many weeks and months and even several years over which the author's cases have been analyzed. But what earthly chance is there of doing this except for the few favored (or unfortunate) persons

who have wealth? And if good results are so delayed and are not always obtained (Mrs. Evans fails to give final outcomes in many instances), it is an open question whether there must not be vastly more direct routes to self control or sublimation, such as punishment, social or physical and even corporal, or through such exigencies as economic necessity for early meeting the world on its own terms by working for a living or through strong moral or religious influences. How otherwise are people working themselves through their difficulties—is it only the well-to-do that do have these sterner chances for conquering themselves or receiving simpler aid?

Indeed, is it not a fact that the great part of the whims and vagaries of behavior, which in this book are apparently offered as the symptoms of "The Nervous Child," are only possible in circumstances of some affluence? There can be little doubt that a highly protected environment gives proportionately more opportunity for unfortunate reactions of any inner urge (libido). The world of her patients, we glean from Mrs. Evans' descriptions, is one of rich velvets and many toys and pleasures for the children, fine jewels or big business interests for the parents. Not that we would maintain that poverty gives freedom from untoward effects of mental conflict—the reviewer has published many cases showing the opposite. But the point is that psychanalysts have too little faced the fact that certain social and economic circumstances largely condition the ailments of their clientele as well as create the possibilities of psychanalytic treatment. The frank statement of some that they will do nothing for those who come from a "lower" social level or who cannot pay well may be in recognition of the immediate cardinal facts, but that makes it possible to draw some interesting practical deductions from two angles.

To form a book best adapted for parents on parental influences and correlated possibilities for good and evil, it seems to the reviewer that a culling process might well be undertaken and the many good paragraphs be separated from the mere fanciful and less surely founded features of the text. The latter will draw the reader's attention, perhaps somewhat morbidly, but, after all, these passages may so embody the author's own credo that she would be unwilling to drop them.

Fifty cents each will be paid for the April and May, 1919, issues of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY. Address to American Medical Association, 535 North Dearborn St., Chicago, Ill.